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THE
LARYNGOSCOPE.

VOL. LXIII MARCH, 1953 No. 3

BRONCHOLOGIC AID IN DIAGNOSIS.*

LOUIS H. CLERF, M.D.,
Philadelphia, Pa.

Bronchoscopy was first perfected for the removal of foreign bodies from the air passages. In performing these procedures considerable information was obtained concerning the physiology of the tracheobronchial tree; in addition, much data bearing on pathological processes was accumulated. Since many of the obscure diagnostic problems in pulmonary diseases simulate the physical and Roentgen findings observed in cases of bronchial foreign bodies, it appeared obvious that the bronchoscopic findings in these would be helpful. This was particularly evident in cases of partial or complete bronchial obstruction and in wheezing respiration of unknown etiology, since these clinical observations commonly were noted in foreign bodies in the air passages.

The growing popularity of bronchologic methods in diagnosis and therapy was reflected in the increasing number of patients that were referred to the various bronchoscopic clinics.

During 1923, when there were but two bronchoscopic clinics in Philadelphia, 842 endoscopic procedures were performed at the Clinic of the Jefferson Hospital. About 12 per cent of these were foreign body cases. During 1932, when 13 bron-

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choscopic clinics were actively functioning in as many hospitals in Philadelphia, 3,545 endoscopic procedures were carried out at the Jefferson Clinic. Approximately 2 per cent of these were foreign bodies.

This increase in the volume of bronchologic work in medical centers over the entire country and the resulting need for a greater number of bronchologists may aid in explaining why surgeons and internists began doing this work in many localities. It also may serve to answer the question, who should do bronchology. There can be no doubt that one of the most important considerations in a discussion of bronchologic aid in diagnosis is the availability of a competent bronchologist.

The early bronchologists were otolaryngologists as the specialty was developed in this branch of medicine. The rapid expansion of the specialty, a paucity of otolaryngologists trained in bronchology, limited facilities for instructing those interested in this field, and the desire of thoracic surgeons and internists to investigate and treat patients in their own fields led to the training of bronchologists in a variety of specialties and in an expansion of the field. It also was responsible for the short courses in bronchoesophagology.

What does one mean by a specialist in medicine? A specialist is a practitioner who devotes himself to a special department of medicine and who has qualified himself by securing special knowledge. It can be assumed, therefore, that a bronchologist is a specialist who is interested in diseases of the tracheobronchial tree and also has been trained to carry out bronchologic procedures, in the broadest sense of this term. The laryngologist who does not manifest an interest in diseases of the tracheobronchial tree is no better qualified to be the bronchologist in a general hospital or a "chest clinic" than is the thoracic surgeon who is familiar with the technical phases of introducing a bronchoscope but is not familiar with the appearances of either the larynx or the lower air passages.

To be a competent bronchologist, one should be able to do bronchoscopy safely. This entails not only a technical knowl-

edge of introducing endoscopic tubes and using forceps, aspirating tubes and other equipment, but also an intimate familiarity with the indications and contraindications for bronchoscopy. In addition, one should be able to do endoscopic procedures with a minimum of trauma to the patient. The occurrence of postbronchoscopic complications too often is an evidence of lack of skill and judgment on the part of the bronchologist. A competent bronchologist also should be familiar with the anatomy, physiology and pathology of the tracheobronchial tree. He should be able to note all abnormalities, be familiar with the performance of proper biopsy, secure secretions from involved pulmonary lobes or segments and be able to interpret for the surgeon or internist the findings that he has observed.

Obviously, it is impossible to indicate who shall do bronchology in a given institution. In large clinics or centers it is imperative that a trained bronchologist is available at all times. The needs of the thoracic surgeon require this and if the otolaryngological department does not have adequate personnel properly trained and available at all times to take care of postoperative surgical complications, as well as other emergencies that may arise in a general hospital, it usually results in a division of the department and diversion of the work. This undoubtedly was, and still is, the most common reason for training bronchologists outside otolaryngologic departments. I have been impressed that, next to competency in bronchology, is availability for bronchoscopy and that this often determines who does the bronchologic work.

I believe that in teaching centers the interests of bronchology and other departments to which it is an ancillary service would be better served if it were under the jurisdiction of otolaryngology. The "surgeon" bronchologist rarely is equipped or trained to take care of cases of foreign bodies in the air passages and commonly is not available to provide adequate diagnostic services for the departments of medicine, pediatrics, obstetrics, or anesthesiology.

The place of bronchology in medicine may be placed under two headings; namely, diagnostic and therapeutic. Since this

presentation is interested in bronchologic aid in diagnosis, this phase will be discussed. It should be obvious that the entire bronchial tree cannot be visualized through a rigid tube and accessory aids as telescopic inserts which permit one to view lesions "around the corner" must be utilized. Some clinicians still express disappointment that peripherally situated pulmonary lesions cannot be visualized endoscopically. Dependence must be placed on flexible tipped aspirating tubes to obtain secretions from bronchial subdivisions that cannot be observed through a rigid tube and often to determine their patency. Although bronchography is not necessarily a bronchologic method of diagnosis in certain instances it becomes a bronchologic problem.

In addition to establishing a diagnosis by direct visualization, by biopsy, or on the basis of a study of secretions, much valuable information can be secured and considered in conjunction with Roentgen and physical findings; namely, the presence of compression, deformity, fixity, rigidity, or stenosis of a part of the tracheobronchial tree. An opinion on the mobility of the larynx is important in bronchogenic and mediastinal neoplasms. This information is of great assistance to the surgeon and internist in planning therapeutic procedures and often it is of prognostic importance.

In recent years the greatest contributions made diagnostically by bronchology lie in the field of bronchogenic neoplasms and tuberculous tracheobronchitis. Since it would be repetitious to consider all the clinical conditions in which bronchology can provide diagnostic assistance, only these will be discussed.

In bronchogenic neoplasm it is not sufficient to secure a biopsy or collect secretions as an aid in diagnosis. Obviously these must be properly performed for a negative opinion or a false positive, due to inadequate biopsy or improper collection of secretions, would seriously interfere with the proper therapeutic disposition of a case. In addition to these, it is important for the bronchologist to express an opinion concerning the entire tracheobronchial tree beginning at the

larynx. Paralysis of a vocal cord, particularly of the left, is a common contraindication to thoracotomy in bronchogenic carcinoma. Any disturbance in the contour of the trachea, widening of the angle of bifurcation of the trachea, interference with free movement of the carina, encroachment on any of the bronchial subdivisions, deformity, fixity or rigidity is important evidence for the surgeon, and it may determine for him whether thoracotomy should be done. In certain cases an opinion on the condition of the esophagus is helpful. For the same reason, evaluation of a case of esophageal carcinoma is incomplete without an opinion on the status of the trachea and left main bronchus. Accurate and complete observations, therefore, are important.

Before securing secretions for cytologic study, the bronchologist should ascertain the anatomical location of the suspected lesion from a lobar standpoint and also should determine if it will be necessary to posture the patient to obtain a representative specimen. An instrumentarium which includes an assortment of straight and curved flexible tipped aspirating tubes and appropriate collectors is necessary. It is our opinion, based on a considerable experience, that bronchial lavage is an important adjunct for collecting secretions for cytologic study. Not infrequently, the success of cytologic diagnosis is in the hands of the bronchologist and not those of the cytologist.

Tuberculous tracheobronchitis is an important field for the bronchologist. In many clinics it is a routine procedure to precede any plan of collapse therapy by diagnostic bronchoscopy. The reason for this is obvious, for in the presence of endobronchial disease producing partial bronchial obstruction collapse therapy may not only be unsuccessful but may also be harmful. It is, therefore, important that the larger bronchi be explored and also that an opinion is given concerning the smaller subdivisions, particularly the upper lobe bronchi, which can be visualized only with a telescopic insert. If obstruction is present it is important to know whether it is due to secretions, infiltration, ulceration, granulation or cicatrization.

The needs for bronchoscopic therapy in tuberculous tracheobronchitis have been replaced largely by the antibiotic agents, but, in the field of diagnosis, bronchologic aid still is important.

Not infrequently the bronchologist is asked to decide whether a patient who exhibits certain unexplained symptoms should be subjected to bronchoscopic investigation. Among these are wheezing respiration, unexplained cough, and hemoptysis.

Wheezing respiration commonly is indicative of partial bronchial obstruction, although it need not necessarily be an inspired foreign body or organic disease, for secretions, too, may produce this symptom. Appreciation of the significance of wheezing and of the medical history will aid materially in reaching a decision.

Unexplained cough may warrant bronchoscopic investigation, but the competent otolaryngologist also will be aware that a certain number of extrarespiratory causes of cough are recognized. In his preliminary examination of the upper air and food passages he may be able to discover that the cause is not of bronchogenic origin.

Hemoptysis is often a difficult symptom to explain. Here, too, a decision must be made by the bronchologist if and when bronchoscopy should be done. As a rule, remarkably little information will be secured unless hemorrhage has been recent, except when there is Roentgen or physical evidence of a pulmonary lesion. The better results are obtained if bronchoscopy is done promptly after bleeding, assuming that the hemorrhage has not been severe. In this way one may trace tell-tale blood-tinged secretion to a lobar or segmental bronchus.

Unexplained Roentgen findings, notably obstructive emphysema or atelectasis, are a fruitful field for bronchologic investigation, as many of these are the result of either endo- or extrabronchial lesions. Endobronchial lesions commonly can be diagnosed by bronchoscopic investigation. Dependence

here rests on an interpretation of the findings, the determination whether biopsy should be done or whether cytologic and bacteriologic examination is indicated. Extrabronchial disease may necessitate bronchography and tomography.

While bronchography is an important diagnostic aid, there are instances when it should not be employed. Among these are cases where comparative Roentgen studies are contemplated or where immediate pulmonary surgery is indicated.

To summarize briefly, bronchologic aid in the diagnosis of bronchopulmonary disease is important. To provide this aid the bronchologist should be properly trained so that he can do bronchoscopy skillfully and with a minimum of trauma. He should be familiar with all the endobronchial aspects of bronchopulmonary disease so that he can provide a comprehensive and intelligible evaluation of the case, and lastly, he should be readily available.

1530 Locust Street.

ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology, divided into two sections:

A. Basic Section, Sept. 21 through 26, 1953, devoted to surgical anatomy and cadaver dissection of the head and neck, and histopathology of the ear, nose and throat, under the direction of Dr. M. F. Snitman.

B. Clinical Section, Sept. 28 through Oct. 3, 1953, consisting of lectures and panel discussions, with group participation of otolaryngological problems and current trends in medical and surgical management.

Registration will be limited. Application for attendance at one or both sections will be optional. For information, write to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

FACTS AND FALLACIES OF BONE CONDUCTION.*

FRANCIS L. WEILLE, M.D., and
SAMUEL GARGANO, B.S., (by invitation),
Boston, Mass.

PART I.

It is the purpose of this paper to clarify and *not* to confuse the mind of the busy clinician in otology, as to some of the useful present-day concepts of bone conduction held by the acoustic psychologist, and to contrast the former's "practical" with the latter's "scientific" view. It is highly desirable for the otologist *not* to become lost in the nebulous mists of research audiology. In such research, experiments are carried out upon animals, or upon trained human volunteers. These laboratory workers are deeply interested in the science of sound, its mathematical equations and graph summations which are often confusing or completely unintelligible to the practitioner who is not a higher mathematician. The aurist is not expected to have a detailed knowledge of electroacoustics such as is possessed by a sound engineer; yet it is a reasonable objective for otologists to make use of the simple basic concepts of psychoacoustic investigators, but such ideas must be stated in understandable medical terms free of complicated mathematical formulae and puzzling curves on intricate charts. The quality of such work must have a genuine scientific basis of unquestionably accurate measurement so far as is possible today, and must always be re-evaluated as medical progress continues.

The two most outstanding contributions in recent years to experimental bone conduction investigations have been made by Békésy[1] and Bárány.[2] Their work makes clear to the clin-

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ician the complicated nature of the events resulting when a bone conduction oscillator is applied to the skull. Their data requires practitioners of otology to study further the interpretation of present-day tuning fork and bone-conduction audiometric tests.

Hirsh[3] has stated that in the Harvard Psychoacoustic Laboratory it is the consensus of opinion at the present time that it is impossible to measure bone conduction thresholds accurately because of the many variables involved. In this laboratory Békésy is senior research Fellow in psychoacoustics.

In contrast, Carhart and Hayes,[4] in a study of 250 patients, concluded that the relative reproducibility of air and bone thresholds possesses similar reliability; furthermore, Carhart[5] is convinced that bone conduction measurements which are clinically adequate can now be obtained. Lierle and Reger[6] lean toward this view. All make reservations, however, regarding room noise, proper masking, special application and calibration of oscillators, alteration of the commercial bone conduction vibrator to the hearing aid or individually constructed type, etc.

In any event, many otologists believe that, regardless of theoretical considerations, invaluable information can be obtained from the usual bone conduction tuning fork and audiometric examinations. Very important decisions are frequently based upon such data. It seems reasonable to assume that empirical facts reviewed in terms of measured experimental data will offer to the practical clinician the opportunity for a broader outlook regarding the problem. The discussion in this paper will be limited to bone conduction audiometry.

VARIABLES IN BONE CONDUCTION.

There are three important mechanisms by which displacements of the basilar membrane occur[1,2] in true bone conduction:

1. *Inertia bone conduction* takes place most importantly at low frequencies, and means movement of the skull relative to the footplate of the stapes, as a result of inertia of the ossicular chain.

Displacement of the basilar membrane then occurs exactly as in air-conducted sounds: that is, at low frequencies, the inertia of the ossicular chain causes a lag in following simple parallel vibrations of the skull in the same direction, and this results in displacements of the ossicles relative to the medial wall of the middle ear. Békésy[1] specifically states that below 200 c.p.s. the vibration pattern of the skull is that of a rigid body. Bárány[2] emphasizes the predominant importance of inertia bone conduction at 435 c.p.s., although at this frequency compression bone conduction is very definite also. In general, the inertia mechanism is a major factor up to about 800 c.p.s.

2. *Compression bone conduction* predominates at high frequencies from about 1,600 c.p.s. Bending of the capsule of the skull compresses the bony labyrinth; the unyielding fluid content displaces the basilar membrane because the membranous round window moves more easily than the oval window containing the stapedial footplate, and because the fluid reservoir in the vestibule and semicircular canals causes greater displacement of the fluid in the scala vestibuli than in the scala tympani. Vibration patterns of the cochlear duct result just as in air-conducted sound.

Expansion has converse effects, the round window being sucked in.

3. *Both mechanisms* are very important in the 800 to 1,600 c.p.s. area, and are probably present in variable degrees as indicated over most of the hearing range.

If the oscillator is shifted to a diametrically opposite point of the head, compression bone conduction retains its phase but changes in amplitude, while inertia bone conduction keeps its amplitude but changes phase.

This all seems simple enough, but inertia conduction must be divided into translatory and rotatory elements, according to the direction in which the skull moves when a force is applied; moreover, the rotation may be about a vertical and a horizontal axis. The position of the oscillator determines the

direction in which the skull moves, and, therefore, the amount of motion of the ossicular chain. Rotatory inertia bone conduction about a horizontal axis has an effect on the ossicular chain proportional to the cosine of the angle between the axis of rotation and the axis of the chain.[2] About a vertical axis, no general statement can be made except that this component may support or counteract other components.

Compression bone conduction is complicated by the fact that large vessels "cushion" the bony labyrinth—the carotid artery anteriorly, and the jugular bulb inferiorly. Bárány is of the opinion that compression decreases when the bone conduction receiver moves away in a horizontal plane from the labyrinth in question; but because the labyrinths are located so that they are placed close to the plane of symmetry of the skull, the variations of distance are not great, and, therefore, the magnitude of compression conduction is fairly constant when the horizontal plane only is considered. Oscillations in a vertical plane require pushing the neck down, and clear visualization of what happens is difficult.

Herzog[7] explained compression conduction by the theory that the round window bulges more easily than the oval window when intralabyrinthine pressure is raised. Other factors can be changes in the volume of the vascular bed and extrusion of endolymph in minute quantity through the ductus endolymphaticus into the saccus endolymphaticus which lies in the posterior fossa; moreover, inertia motions of the condyles of the jaw may give rise to compression of the labyrinths.

Air-conducted sound may be generated in the mastoid cells or middle ear air spaces by a bone conduction oscillator. The receiver may also put the auricle or cartilage of the external canal in motion and thereby produce sound waves in the air.

Air-pressure tension in the middle ear can affect bone conduction, as is well known.

The fact that the soft tissues between the bone conduction receiver and skull play an important part in bone conduction

measurements has long been understood. This influence is least when the firmness of application of the oscillator is high. Bárány[2] states, "It seems impossible to design a *single* bone conduction receiver whose loudness is in some measure independent of the condition of the soft tissues when used for a fairly large range of frequencies." With an average firmness of pressure of ±50 per cent, there is a variability in the transmitted tone of less than 5 db for low frequencies and of about 10 db for high frequencies, depending on the construction of the oscillator. If the receiver is not flatly applied to the skull, another error is introduced. This has been measured as 2 per cent for an inclination of 10 degrees, 7 per cent for 20 degrees, etc. Another possible source of error is an air tone emitted by the vibrating skull.[2]

Bone conduction is sometimes greater than when the receiver is applied to the opposite ear; moreover, bone conduction may disappear entirely at points on the head of a person known to have normal ears. The oscillator may emit an air tone audible at certain frequencies and thereby cause error in bone conduction measurements. Its ability to set the soft tissues of the external canal into vibration chiefly through "cartilage conduction" has been postulated and actually measured by Bárány.[2]

Many other complicating factors could be mentioned, but the foregoing briefly summarizes a few of the reasons for checking the validity of bone conduction determinations. The most obvious conclusion for the clinician is that masking must be routinely employed for the ear not being examined, since both ears are always stimulated, and there is no "drop through the skull" of sound pressure in inertia bone conduction. In compression conduction a "drop through the skull" does occur.

MASKING.

It may be helpful in this discussion to summarize some of the present-day quantitative concepts of using *air-conducted* sound to mask one ear while the other is undergoing bone-conduction threshold tests.

1. A white noise masking device with a wide (100 c.p.s. to 10,000 c.p.s.) and as nearly a flat amplification curve as possible is the instrument of choice to deliver the sound emitted from the earphone to the masked ear. For practical purposes, the sawtooth noise built into some audiometers (because it is simpler and less expensive to construct) is acceptable.

2. The latter has its zero setting at the "just audible" level. The former, starting from *no* sound-pressure, has its "zero" about 15 to 20 db *lower* than sawtooth noise. This point is very important, as will be seen later.

3. Masking simply alters the threshold of the ear to which it is applied. It does not "blot out" all hearing in that ear by making *all* nerve fibres refractory at the same time, even at very high sound-pressure levels; but high masking sound-pressure *can* mask the opposite ear somewhat (see below).

4. A white noise sound pressure starting from *nothing* must reach approximately 30 db in *either* ear before any threshold is affected. Above this level, the threshold is altered decibel for decibel.[8]

5. About 50 db are required for sound to go around the head to the opposite ear *if it is covered by a well-fitting earphone.* Since the earphone on that ear is *not* in use in bone-conduction threshold tests, this subtracts about 20 db — the sound excluded by air because of the presence of an earphone on the unmasked ear—reducing this factor to approximately 30 db. By adding these figures to that mentioned in paragraph 4, a minimal *masking* sound of about 60 db on the ear *not* being tested is allowable in all bone-conduction scores if both ears are "normal." If the ear undergoing threshold test is deaf to air-conducted sound, the amount of this deafness in decibels may be added to the masking sound if the examiner so desires.

6. The air-conduction receiver on the masked ear begins to generate bone conducted sound at about 60 db. If 30 db allowable noise *not* affecting threshold is added to the 60 db, it is

concluded that shadow bone-conduction masking of the ear under threshold testing can be excluded, since *air-conducted sound around the skull* will always "get there first."

In the curves to be described, as will be seen from the different zero levels mentioned above, the E-2 Maico sawtooth noise at 100 db is equivalent to the white noise at about 115 to 120 db, *i.e.*, in some of the curves the masking affected the ear under test to the extent of about 55 db. If this ear were "normal," its threshold would be altered to about this amount.

7. The foregoing applies to *bone-conduction* thresholds, not *air-conduction* audiograms where earphones are applied to both ears. In the latter curves, the ear under test does not require masking of the opposite side if the difference between the two sides is less than 40 to 50 db — the minimal s.p.l. need for air-conducted sound to reach the other side; but if the "good ear" is to be masked, one can start with 70 to 80 db white noise or 50 to 60 db sawtooth noise (where the zero is "just audible," as mentioned in describing these two types of masking). The only difference between air and bone conduction masking is the 20 db additional factor of safety in the covering of both ears with well-fitting ear phones held by a headband.

Ambient noise in the examining room of the cases to be discussed was 43 db at its *maximum,* as determined by a noise level meter. This resulted in a 13 db or less masking effect, since the first 30 db has no effect on thresholds.

PART II.

CLINICAL EVALUATION OF EXPERIMENTAL BONE CONDUCTION CONCEPTS. FOUR CASES.

Much information of practical interest to otologists might be obtained if it were possible to eliminate masking entirely, as well as many of the errors of clinical bone conduction tests, by examining the "bad" ear in several patients totally deaf on one side and having practical or essentially normal hearing

on the other side. This would allow a simple method of setting up "shadow" curves on the deaf side, the avoidance of "cartilage conduction," the use of the mastoid process on the deaf side in placement of the oscillator but with elimination of the errors[2] of so doing, minimizing of any air tones, and most important, an estimate as to the levels of inertia and compression elements in cross hearing. That is, the oscillator could be placed 180 degrees from the functioning single ear, or anywhere on the skull, with assurance that there is only one ear functioning.

Of much practical value to the *average* aurist also would be the opportunity to determine the test-retest bone conduction results with two different audiometers and their commercial oscillators. It is pertinent that the vast majority of otologists, and ear clinics, do not possess other than the usual commercial equipment, and must rely upon it for their measurements; moreover, our apparatus was known to be well calibrated. A sound engineer with years of experience in the Harvard Psychoacoustic Laboratory checked the examining room and found its acoustic properties and noise level to be very satisfactory.

It was possible for the authors to carry out observations repeatedly in a number of persons who had practical or essentially normal hearing on one side, with total surgical destruction of the function of the membranous labyrinth on the other side, carried out in the treatment of Ménière's syndrome. Observations on the first four patients so operated upon are included in this paper. Almost total unilateral deafness had been present in each case, but the deaf ear had been found, on appropriate tests, to be the source of the attacks of prostrating vertigo.

In each patient, therefore, multiple bone conduction tests were done with and without masking; moreover, two different audiometers, Maico E-2 and Western Electric 2-A, were utilized so that different type oscillators could be evaluated and the results compared.

Case 1: The hearing status of this 40-year-old white female's right ear was essentially normal, and this ear will be referred to as the normal side.

If the averages of nine air-conduction tests (four Western Electric 2-A and five Maico E-2) are combined into a "composite" audiogram, the following is the result:

Frequency	64	125	250	500	1000	2000	4000	8000
	10	10	10	10	5	10	5	15

A spondee speech audiogram gave a loss of 15 db for this ear. The phonetically balanced word score at 100 db s.p.l. was 86 per cent.

The left ear underwent destruction of the membranous vestibular labyrinth, following which there was no hearing by air conduction if the normal ear was masked by white noise at 100 db s.p.l. or sawtooth noise at 90 to 100 db.

Phonetically-balanced word tests gave no response at 115 db s.p.l. if the normal ear was masked. The finding was present both before and after operation; therefore, for practical purposes, the left ear was "dead," and the right ear within normal limits.

SHADOW CURVES IN AIR CONDUCTION TESTS.

A typical unmasked *air-conduction* audiogram (Maico E-2) was as follows:

Frequency	125	250	500	1000	2000	4000	8000
Normal Ear	15	5	5	0	0	+5	25
Dead Ear	70	70	65	75	65	65	60
Difference	55	65	60	75	65	70	35

With masking of the normal ear at 100 db sawtooth noise there was no response in the dead ear.

Is this difference caused by *bone-conduction through the skull,* or by air conduction *around* the skull? From what has already been said, it is obvious that only one ear is involved in the responses, the other being "dead."

It is expected that by air conduction *around* the head, a sound pressure of about 30 db in one ear will reach (through the headband?) the opposite side.[8] A well-fitting earphone will exclude about 20 db of sound, so that approximately 50 db are required to reach the ear itself; but up to 4000 cycles, 5 to 25 db *more* than the minimal 50 db were required for a response.

By means of skull electrodes, Békésy has shown that when an *earphone* reaches a 60 db output, the skull begins to vi-

brate; that is, bone conduction starts. It is likely, therefore, that at 8000 cycles, an *air* conduction mechanism was at work, since only 35 db s.p.l. was required to produce a shadow response, but at the other levels one or both stimuli may have been important.

SHADOW CURVES BY BONE CONDUCTION.

The following unmasked bone conduction audiogram was made before removing the left membranous labyrinth. The figures indicate the "loss" in decibels:
W.E. 2-A:

Prior to destruction of left labyrinth:

Frequency	64	128	256	512	1024	2048	4096
Normal Ear	10	15	15	20	20	15	10
Deaf Ear	10	15	30	30	25	20	20

With masking at 100 db white noise on the normal side, there was no response from the deaf ear.

After the operation repeating the test with the same audiometer gave the following figures:

One month postoperative:

Frequency	64	128	256	512	1024	2048	4096
Normal Ear	5	15	30	25	30	25	10
Deaf Ear	10	10	15	40	40	30	20
Difference	—5	+5	+15	—15	—10	—5	—10

Three months postoperative.

Frequency	64	128	256	512	1024	2048	4096
Normal Ear	5	10	10	5	10	5	0
Deaf Ear	—	—	15	15	20	20	25
Difference	—	—	5	10	10	15	25

Four and one-half months postoperative.

Frequency	64	128	256	512	1024	2048	4096
Normal Ear	10	15	10	10	5	5	15
Deaf Ear	10	15	20	25	15	20	25
Difference	0	0	—10	—15	—10	—15	—10

The effect of masking the normal ear at 70 to 100 db was to abolish the responses in the deaf ear except for occasional perception of a feeling of vibration in the lower frequencies.

The following postoperative bone conduction audiograms without masking were made about two to six weeks apart on the Maico 2-E instrument:

Frequency	125	250	500	1000	2000	4000
Normal Ear	30	40	35	45	25	20
Deaf Ear	25	40	35	20	20	35
Difference	+5	0	0	+25	+5	—15
Normal Ear	30	40	40	55	20	20
Deaf Ear	—	15	20	20	20	25
Difference	—	+25	+20	+25	0	—5
Normal Ear	30	35	35	15	5	0
Deaf Ear	30	25	25	15	20	15
Difference	0	+10	+10	0	—15	—15
Normal Ear	30	30	25	30	10	5
Deaf Ear	—	—	30	20	20	35
Difference	—	—	—5	+10	—10	—30
Normal Ear	30	35	30	20	10	5
Deaf Ear	—	35	40	20	25	25
Difference	—	0	—10	0	—15	—20
Normal Ear	30	30	30	10	15	15
Deaf Ear	—	30	30	15	25	20
Difference	—	0	0	—5	—10	—5
Normal Ear	30	35	25	50	35	20
Deaf Ear	30	35	30	20	20	30
Difference	0	0	—5	+30	+15	—10
Normal Ear	30	20	10	5	+5	+5
Deaf Ear	20	10	5	5	0	+5
Difference	+10	+10	+5	0	—5	0

Chart I summarizes the shadow hearing curves in the dead ear:

CHART I.

Case 1: Shadow hearing: dead ear responses. 8 Maico E-2 B.C. audiograms. Tests two to six weeks apart.

PREDOMINANTLY

Inertia B.C. 500 c.p.s. or less			Mixed 1000 c.p.s.		Compression B.C. 2000-4000 c.p.s.	
Dead ear response	Times	%	Times	%	Times	%
Same	8	66%	3	87%	2	25%
Better	8		4		2	
Worse	8	33%	1	12%	12	75%

This indicates that inertia bone conduction is very important in these tests at 1,000 cycles or lower, while compression conduction predominates at higher frequencies, and that a "drop through the skull" in sound pressure is implied by the responses at 2,000 to 4,000 c.p.s. Both mechanisms seem to be present throughout the scale in these measurements.

Variability in these eight audiograms:

Frequency	125	250	500	1000	2000	4000
Normal ear	0 db	20	30	50	40	25
Deaf ear	10 db	30	35	15	25	40
Limits found:						
Normal ear	30-30	20-40	10-40	5-55	+5-35	+5-20
Deaf ear	20-30	10-40	5-40	5-20	0-25	+5-35

The variability and range of the 12 tests in relation to the power limits of the two audiometers will be seen at a glance in Charts II to V.

CHART II.

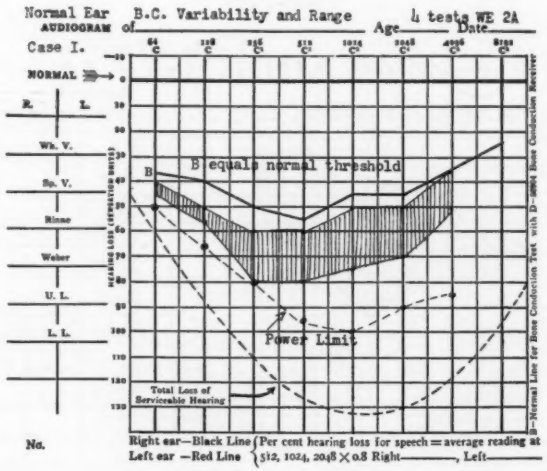


CHART III.

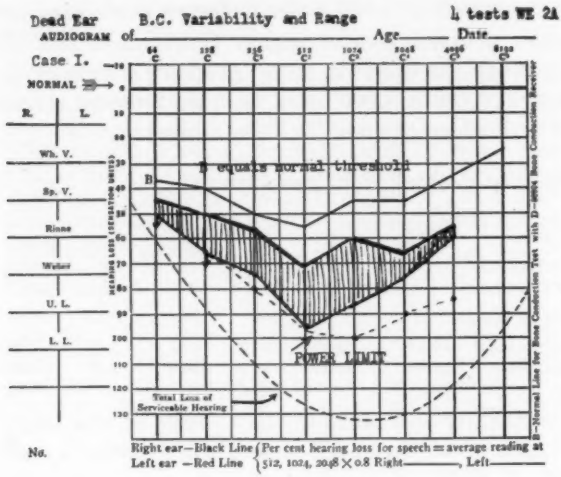


CHART IV.

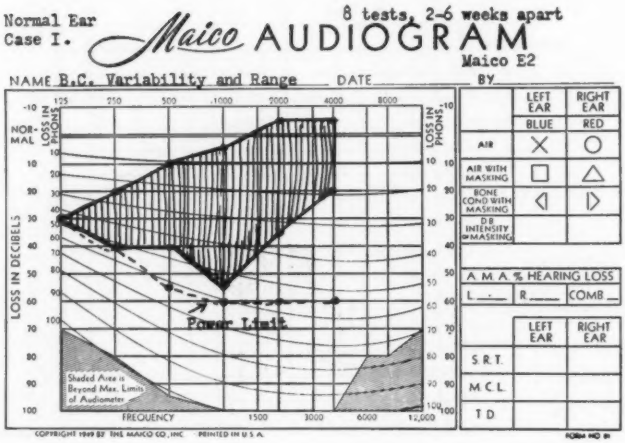
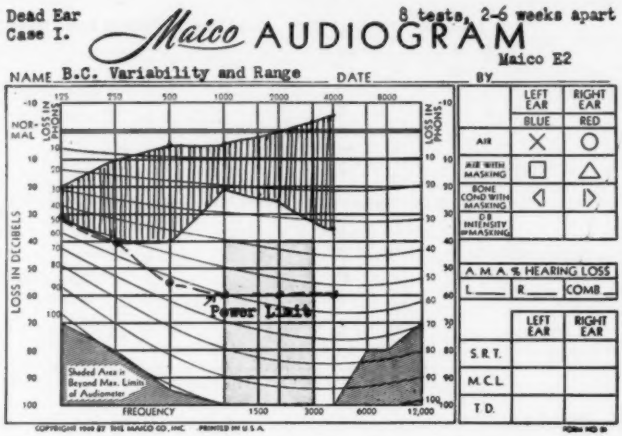


CHART V.



Case 2: Fifty-one-year-old white male. A typical example of the air conduction responses of 24 audiograms (11 W.E. 2-A, 13 Maico E-2) was as follows:

	64	128	256	512	1024	2048	4096	8192
Right Ear	5	5	5	10	5	10	55	40

Left Ear: No response with right ear masked. Highest phonetically balanced word test score at 100 db s.p.l.: 92 per cent in right ear; no response on the left side.

In this patient the left membranous labyrinth underwent fenestration destruction with "cure" of vertigo resulting over a 20-month follow-up.

SHADOW CURVES.

Unmasked bone conduction audiograms postoperatively were done with the W.E. 2-A and Maico E-2 instruments. It will be interesting to present the results by considering the measurements at 500 cycles or less; then at 1,000 c.p.s.; and finally at 2,000 to 4,000 c.p.s.

1. Inertia Conduction: 500 c.p.s. or less.

	Audiogram Number	W.E. 2-A		Maico E-2	
		Normal Ear	Deaf Ear	Normal Ear	Deaf Ear
64 cycles	1	12	7	—	—
	2	12	12	—	—
	3	2	2	—	—
	4	2	7	—	—
	5	+3	7	—	—
		—	—	—	—
Average		5	7	—	—
128 or 125 cycles	1	15	10	20	20
	2	10	15	10	10
	3	0	0	15	10
	4	0	0	20	15
	5	0	15		
		—	—	—	—
Average		5	8	16	14
256 or 250 cycles	1	20	15	10	20
	2	15	25	10	15
	3	+5	0	15	5
	4	0	5	5	0
	5	+5	10		
		—	—	—	—
Average		5	11	8	10
500 or 512 cycles	1	25	10	15	15
	2	25	15	10	5
	3	10	0	5	5
	4	10	10	10	5
	5	20	10		
		—	—	—	—
Average		16	9	10	8

Combined inertia and compression bone conduction at 1,000 c.p.s.:

	Audiogram Number	Normal Ear	Deaf Ear
W.E. 2-A	1	20	45
	2	45	20
	3	10	20
	4	30	30
	5	15	40
Maico E-2	1	20	15
	2	15	10
	3	5	15
	4	5	15

Compression bone conduction measurement:

	Audiogram Number	W.E. 2-A Normal Ear	W.E. 2-A Deaf Ear	Maico 2-E Normal Ear	Maico 2-E Deaf Ear
2000 or 2048 cycles	1	20	40	15	25
	2	20	30	0	20
	3	20	20	10	25
	4	15	20	+5	5
	5	10	20		
Average		17	26	5	19
4000 or 4096 cycles	1	40	50	35	55
	2	40	50	35	40
	3	40	—	35	50
	4	45	45	15	35
	5	50	50		
Average		43	49	30	45

The difference ("drop through the skull") between the two sides varied from 0 to 20 db at 2,000 c.p.s., and at 4,000 c.p.s. In the writer's experience this is the first concrete evidence of a genuine "drop through the skull" in *clinical* bone conduction audiometry.

Chart VI summarizes the shadow hearing results in this case, while Chart VII points out the variability of the test-retest measurements.

CHART VI.

Dead ear responses, *compared to normal ear.*

Case 2: Male, 51 years. Right ear normal. Left ear destroyed. Nine B.C. audiograms; five W.E. 2-A; four Maico E-2.

PREDOMINANTLY

Dead ear response	Inertia B.C. 500 c.p.s. or less Times	%	Mixed 1000 c.p.s. Times	%	Compression B.C. 2000-4000 c.p.s. Times	%
Same	9	69%	1	44%	0	0%
Better	13		3		3	17%
Worse	10	31%	5	56%	15	83%

Variability in the measurements can be summarized as follows:

CHART VII.

Case 2: Nine tests; five W.E. 2-A; four Maico 2-E.

VARIABILITY

c.p.s.	Normal Ear		Dead Ear
64	15		10
125-128	15		15
250-256	25		15
500-512	15		15
1024	35	(W.E. 2-A)	25
1000	15	(Maico E-2)	5
2048	10	(W.E. 2-A)	20
2000	20	(Maico E-2)	20
4096	10	(W.E. 2-A)	5 to limit
4000	20	(Maico E-2)	20

Case 3: White male, 69 years. The following is a composite of 14 unmasked air-conduction audiograms (eight W.E. 2-A; six Maico E-2) of the right ear.

64	125	250	500	1000	2000	4000	8000
25	20	20	25	40	45	65	70

P.B. score: 70 per cent at 100 db s.p.l.

There was almost no hearing in the deaf left ear with masking of the good ear prior to operation, and none postoperatively. The P.B. score of the deaf ear was: 0 per cent at 100 db s.p.l.

INERTIA BONE CONDUCTION SCORES.

		500 c.p.s or less.			
		W.E. 2-A		Maico E-2	
		Good Ear	Deaf Ear	Good Ear	Deaf Ear
64 cycles	1	2	—		
	2	7	2		
	3	2	—		
128 or 125 cycles	1	5	15	20	35
	2	5	15	25	30
	3	0	—	25	35
256 or 250 cycles	1	5	25	25	20
	2	5	25	25	25
	3	—5	20	25	35
512 or 500 cycles	1	30	40	30	35
	2	20	30	35	30
	3	15	30	30	30

COMBINED INERTIA AND COMPRESSION B.C. AT 1000 C.P.S.

	W.E. 2-A		Maico E-2	
	Good Ear	Deaf Ear	Good Ear	Deaf Ear
1	30	50	40	35
2	50	—	40	40
3	30	30	40	45

COMPRESSION BONE CONDUCTION.

		W.E. 2-A		Maico E-2	
		Good Ear	Deaf Ear	Good Ear	Deaf Ear
2048 or 2000 c.p.s.	1	25	—	45	55
	2	35	—	40	—
	3	30	40	40	55
4096 or 4000 c.p.s.	1	—	—	55	—
	2	50	—	50	—
	3	—	—	50	—

At 2,000 to 4,000 cycles a 10 to 15 db "drop through the skull" was demonstrated, and in no instance was the shadow response to or greater than that of the good ear.

In this case there was a 20 to 25 db depression in the air conduction threshold of the good ear below 500 cycles. The question is raised as to the effect which this may have on shadow curves, and points toward the possibility that the inertia effect is greater if there is nearly normal hearing on one side.

The measurements cited are summarized in Charts VIII and IX.

CHART VIII.

CASE 3.

EFFECT OF DEPRESSED A.C. HEARING CURVE UPON THREE TYPES OF BONE CONDUCTION—

Six B.C. Audiograms: Three W.E. 2-A; Three Maico E-2.

Dead ear responses compared to good ear:

PREDOMINANTLY

	Inertia B.C. 500 c.p.s. or less		Mixed B.C. 1000 c.p.s.		Compression B.C. 2000 to 4000 c.p.s.	
Response Dead Ear	Times	%	Times	%	Times	%
Same	2	24	2	50	0	0
Better	3		1		0	0
Worse	16	76	3	50	10	10

CHART IX.

Case 3: Variability. Six tests: three W.E. 2-A, three Maico E-2.

	Functional Ear		Dead Ear	
C.P.S.	W.E. 2-A	Maico E-2	W.E. 2-A	Maico E-2
64	5	—	12+	—
125 to 128	5	5	10+	5
250 to 256	10	0	5	15
500 to 512	15	5	10	5
1000 to 1024	20	0	25+	10
2000 to 2048	10	5	5+	5+
4000 to 4096	2.5+	5	—	—

Case 4: Female, age 60 years. Fenestration destruction of the right ear. Two typical air-conduction audiograms (of 12 done) for the normal (left) ear were as follows:

Frequency	64	125 128	250 256	500 512	1000 1024	2000 2048	4000 4096	8000 8192
W.E. 2-A	0	0	5	5	0	20	20	20
Maico E-2		10	10	10	5	20	20	10
Composite	0	5	5	5	5	20	20	15

The deaf ear responded before operation when the normal ear was masked at 100 db sawtooth noise:

250	500	1000	2000	4000	8000
80	80	100	100	90	80

That is, the right ear was profoundly deaf prior to fenestration.

A spondee speech test of the normal ear measured 11 db loss, and the best PB score at 100 db s.p.l. was 90 per cent on this side. There was an 0 per cent response on the deaf side.

Bone conduction audiometric measurements were as follows:

INERTIA BONE CONDUCTION, 500 C.P.S. OR LESS.

		W.E. 2-A		Maico E-2	
		Normal Ear	Dead Ear	Normal Ear	Dead Ear
64 c.p.s.		5	—		
125 or 128 c.p.s.	1	5	—	45	20
	2			35	35
	3			35	15
	4			25	25
	5			35	—

INERTIA BONE CONDUCTION, 500 C.P.S. OR LESS (Continued).

		W.E. 2-A		Maico E-2	
		Normal Ear	Dead Ear	Normal Ear	Dead Ear
250 or 256 c.p.s.	1	15	30	45	20
	2			35	25
	3			55	15
	4			30	15
	5			35	—
500 or 512 c.p.s.	1	20	35	30	25
	2			30	25
	3			55	0
				20	25
				35	45

MIXED BONE CONDUCTION—1000 C.P.S.

		W.E. 2-A		Maico E-2	
		Normal Ear	Dead Ear	Normal Ear	Dead Ear
1000 c.p.s.	1	20	20	25	10
	2			25	10
	3			45	15
	4			20	20
	5			25	40

COMPRESSION BONE CONDUCTION.

2000 or 2048 c.p.s.	1	20	40	50	35
	2			35	35
	3			45	35
	4			35	35
	5			25	45
4000 or 4096 c.p.s.	1	30	50	45	45
	2			25	35
				35	45
				20	35
				25	55

These results are summarized for shadow curves and for variability in Charts X and XI.

CHART X.

CASE 4.

Female, 60 years. Left ear normal; right ear destroyed. Dead ear responses compared to normal ear—6 B.C. audiograms: one W.E. 2-A; five Maico E-2.

PREDOMINANTLY

	Inertia B.C. 500 c.p.s. or less		Mixed 1000 c.p.s.		Compression B.C. 2000 to 4000 c.p.s.	
Dead Ear Response	Times	%	Times	%	Times	%
Same	2	58	2	83	3	42
Better	9		3		2	
Worse	8		1	17	7	58

CHART XI.

Case 4: Variability—five tests, Maico E-2.

	Normal Ear	Dead Ear
125	20	20
250	25	10
500	35	45
1000	25	30
2000	25	10
4000	25	20

PART III.

DISCUSSION.

1. Types of bone conduction in relation to Békésy's theory have clinical substantiation in this study. In Cases 1, 2 and 4, one ear was essentially normal, the other "dead" so far as function was concerned. In Case 3 there was mixed deafness in the functioning ear of intermediate degree. In all cases an attempt was made to set up shadow curves in the dead ear; these invariably disappeared when the functioning ear was properly masked.

INERTIA CONDUCTION.

Chart I is of particular interest because the measurements at 500 cycles or less for predominantly inertia conduction demonstrate the shadow curve for the dead ear to be better, worse and equal to the normal side exactly the same number of times. Chart VI shows the shadow curve to be the same or better than the normal side 69 per cent of the times for predominantly inertia conduction. This again is in line with Békésy's ideas, and against the almost universal belief that there is a "drop through the skull" of perhaps 10 db for low-tone sound pressures in accordance with the inverse square law principle applied elsewhere. It must be emphasized that inertia conduction postulates simultaneous stimulation of *both* ears because of skull movement roughly toward one set of ossicles and away from the other, the ossicular "lag" being due to inertia affecting one ossicular chain about the same as the other.

In Case 4 the shadow curve is distributed about as would be expected: better nine times, worse eight, and the same twice, as compared with the normal side.

It is obvious, therefore, in these three patients with one ear *normal* and the other totally destroyed, that the hearing ear is stimulated (as Békésy's theory would claim) equally easily from either mastoid region. This seriously challenges the validity of unmasked audiograms at 500 cycles or less if one ear is normal and the other under test.

In Case 3 there was present an intermediate degree of mixed deafness. Although the air conduction curve slanted from left to right, a 70 per cent phonetically balanced speech audiogram score ruled out serious nerve deafness. (The normal has been reported by one of us[9] as 88 per cent, with 82 per cent the lower limit of normal.)

In this patient the shadow curve was, at 500 cycles or less, the same or better than the measurements of the functioning ear only 24 percent of the time. Partial stapes fixation might be the explanation, but a few observations in a single case lend themselves merely to conjecture.

COMPRESSION CONDUCTION.

The shadow curves in the 2,000 to 4,000 c.p.s. area were invariably worse—58 to 100 per cent of the time—than the measurements for the functioning ear. A variable "drop through the skull" of up to 20 db was found frequently, but because both compression and inertia bone conduction are probably present throughout most of the audiometric scale, one can never rely upon a sound pressure "drop through the skull" as an excuse for omitting masking.

VARIABILITY OF BONE CONDUCTION AUDIOGRAMS.

Test-retest variability of bone conduction audiograms is discussed with the hope that our results will stimulate basic improvements in methods, so far as possible. Two factors can be excluded from the clinicians' viewpoint in our findings:

1. masking, since one ear was "dead" in each case; *2.* ambient noise, since this was well controlled in our examining room, as previously mentioned.

With these influences excluded, to have in Case 1, for example, in the important "speech range" octaves a fluctuation at 500 c.p.s. of 10 to 40 db; at 1,000 c.p.s. of 5 to 55 db; at 2,000 c.p.s. of + 5 to —35 db, respectively, will emphasize the basic inaccuracies of the method. It is our belief that the many possible unmeasurable factors outlined earlier in this paper combined to produce our test-retest results. This view requires further discussion as follows:

1. The threshold reference line for "normal" varies from one audiometer to another. This line is meaningless as a reliable reference because no normal has ever been agreed upon by the Bureau of Standards or other authorities. Békésy's work gives an inkling as to why.

CHART XII.

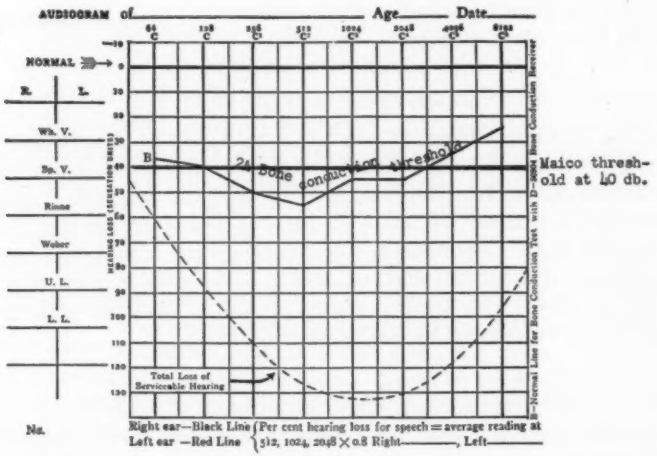


Chart XII points out the differences (up to 15 db) in the "normals" of the two audiometers used in this study. As

spokesman for the Maico engineers, Watson[10] has stated: "We believe that the bone conduction thresholds used with the old 2-A battery-operated audiometer came very close to being as accurate and acceptable as possible.

"The calibration of Maico audiometers is comparable to that of the 2-A battery-operated audiometer."

He goes on to say: "If any audiometer is so calibrated that a normal ear, open or occluded, or a conductively deafened ear can hear far better by bone conduction than any normal ear by air conduction on the same audiometer, the bone conduction calibration is ridiculously unrealistic and false.

"And yet this is precisely what has been done in setting the normal or zero threshold for bone conduction for some audiometers." He mentions that when air and bone conduction are compared in such an instrument at 40 db above threshold, on an equal loudness basis, bone conduction will be favored by as much as 30 db.

2. The oscillator. Bárány[2] has stated that it is impossible to design a wide-band frequency oscillator which will not vary about 10 db over a part of the scale; moreover, the Bureau of Standards has never accepted an artificial mastoid for calibration purposes, nor has any other official body.

Carhart[4] has leaned toward the use of a hearing-aid type of bone-conduction vibrator, and believes[5] that at the present time the user, not the manufacturer, of the unit must calibrate it. He describes methods for so doing. This is certainly a challenge to the makers of commercial units.

3. Firm and even contact of the oscillator with the skull. We believe this to be of major importance. Present-day commercial devices for accomplishing this purpose are of very poor quality, and improvement here should not be difficult. Favorable changes in bone-conduction test-retest scores might follow.

4. Air-tone of the oscillator. It is well known that an air tone is emitted by a bone conduction oscillator. The following experiment is, therefore, of interest:

Using the E-2 Maico audiometer for testing a 53-year-old male having essentially normal hearing, a bone conduction audiogram was done in the usual manner; it was then repeated with the oscillator in the same position but not touching the skin. A third measurement was made with the oscillator lying on a large folded handkerchief upon a table about 12 inches from the ear. The following results were obtained:

1. With the Maico E-2:

Frequency	125	250	500	1000	2000	4000
Oscillator on mastoid	20	15	10	10	10	10
Just off mastoid bone conduction scale	—	35	20	15	30	20
On table	—	—	20	20	35	25

The air-tone persisted with firm pressure of one's thumb on the oscillator.

2. With the Western Electric 2-A:

Frequency	64	128	256	512	1024	2048	4096
Oscillator on mastoid	0	5	0	7.5	2.5	0	2.5
Just off mastoid	—	—	—	32.5	27.5	2.5	2.5

The air-tone disappeared with firm pressure of one's thumb on the oscillator.

Another subject (female, 37 years) with normal hearing listened to these oscillators held six inches from the ear with the following responses:

Maico E-2	125	250	500	1000	2000	4000
(Bone conduction scale)	—	—	20	15	10	0
W.E. 2-A	64	128	256	512	1024	2048	4096
(Bone conduction scale)	—	—	25	30	20	5	+2.5

It is, therefore, obvious that the air tone of the bone-conduction oscillator may sometimes be a factor of error if the subject's air-conduction hearing curve is not sufficiently depressed to neutralize such an effect.

5. "Standard deviations" reported for bone conduction acuity in the National Health Survey[11] varied from 7.4 to 8.7 db.

That is, the "range" was 14.8 to 17.4 db *in approximately the middle two-thirds* of a group of 1,242 individuals. Western Electric 2-A audiometer equipment was used.

It is of interest in Case 1 that with this audiometer the "range" of test-retest variation exceeded 20 db only at 1,024 c.p.s. (with a fluctuation here of 25 db).

PART IV.

What can be done by clinical methods for greater accuracy?

1. Multiple threshold tests in the same patient are the most practical answer with present-day equipment and limitations. As in speech audiometry,[9] the *best* scores are the most informative. Note the *upper* margin of the graph in Charts II and IV.

2. Phonetically balanced speech audiometric scores[9] at high sound-pressure levels offer a very simple, rapid, and with multiple repetition, reasonably accurate method of checking the validity of any bone-conduction audiogram.

3. Possibly *masked* above-threshold bone-conduction audiograms. The method described by Watson[12] is not accurate because no masking is used, and it is, therefore, impossible to know which ear is responding. The controls already present in the Maico E-2 audiometer permit in this test approximately 55 db of masking noise, or about 25 db depression of the hearing threshold for the normal ear utilized in the equivalent of binaural loudness balance, the air-conducted pure tones being introduced into the normal ear alternately with bone conduction stimulation of the deaf ear while the normal ear is masked. In perceptive deafness cases, the possibility of recruitment as a factor of error would have to be considered in trying to use this method.

4. The avoidance of overmasking is important. Overmasking may be defined as depressing the threshold of the ear being tested by white or sawtooth noise introduced into the opposite ear to avoid cross-hearing.

As previously stated, it has been emphasized experimentally[8] that the first 30 db of white noise arriving at *either* ear will not affect threshold measurements. The discussion regarding masking earlier in this paper will allow the reader by simple calculations to determine the risks of overmasking.

In our cases, deliberate overmasking was sometimes carried out in an attempt to estimate the drop in sound pressure "around the skull," since it is our impression that this varies (for low or high tones or both) from one test to another, depending upon the way the earphone and headband are applied to the masked ear, and other factors.

By subtracting the threshold measurement plus 30 db from the sound pressure of the masking noise at the opposite ear, a theoretical calculation of the "around the head" drop can be made.

For example, in Case 1 by masking the functionally dead ear at 100 db sawtooth noise (equivalent to about 115 db of white noise), the following bone conduction curve was obtained for the normal ear:

Frequency	125	250	500	1000	2000	4000
Threshold	40	40	45	40	40	60

If each threshold measurement has added to it the ineffective first 30 db arriving by air, and the resulting figure is subtracted from 115 db, the "around the head" drop in sound pressure would be:

Frequency	125	250	500	1000	2000	4000
Db	45	45	40	45	45	25

Average: 40 db.

If the bone conduction determinations were reliable here, one would assume that the drop around the head may vary from octave to octave, and that the assumption of about 30 db mentioned before is conservative.

A repetition of the test in the same case was carried out at 70 db sawtooth masking, equivalent to about 85 db of white noise. By calculating 85 db — (30 db + 30 db) = 25 db, one would expect to find overmasking in this amount—namely, 25 db.

The actual figures obtained were:

Frequency	125	250	500	1000	2000	4000
Threshold	20	30	30	15	25	15

Average: 22.5 db.

It must be pointed out that after reaching a sound pressure of about 30 db, the *masked* ear has its threshold depressed decibel for decibel. Thus, if 90 db of white noise were used, the threshold of this ear should be depressed approximately 60 db. This would equal the maximum range of power attainable for a pure tone bone-conduction curve on the opposite side in the audiometers used by us. These power-limits for each frequency have been shown in Charts II through V.

CONCLUSIONS.

1. Otologists must seriously evaluate for clinical use some of the present-day concepts of bone conduction held by acoustic psychologists.

2. Békésy's theories of bone conduction receive support from clinical studies of the four patients reviewed here. The fact that each of these patients had one "dead" ear functionally made such an investigation possible.

3. The *hypothesis* that inertia bone conduction stimulates *both* ears simultaneously by motion of the skull relative to the lag of the ossicles by inertia means approximately equal stimulation bilaterally insofar as this particular mechanism is concerned.

4. While inertia bone conduction is believed to be most important at low frequencies, and compression conduction at high frequencies, both are theoretically present throughout most of the audiometric scale; therefore *no unmasked bone conduction audiogram is valid if both ears are functioning* because of uncertainty as to which ear is responding to the test tone.

5. Quantitative masking, as outlined, will prevent overmasking and will inform the examiner as to the approximate alteration in the threshold of the masked ear.

The most useful single figure cited in this respect is that approximately *the first 30 db of noise heard in either ear has no effect upon thresholds* if the noise zero equals 0 sound pressure.

6. Békésy's fundamental concepts apply equally to tuning forks used as bone oscillators if his theory of bone conduction is correct.

7. The presence or absence of compression bone conduction probably explains the presence or absence of a "drop through the skull" in sound pressure in bone conduction audiograms.

8. Variability in bone conduction test-retest audiometric results is controversial as to magnitude. The *best* scores of multiple determinations over a period of time are the probable index of true function. Repeated phonetically balanced speech audiograms carried out at a high sound pressure level (usually about 100 db)offer a means for checking the validity of bone conduction measurements.

9. At the present time there are no widely accepted standards for calibration of the bone conduction zero reference level built into audiometers; therefore, substantial variations may occur from one make to another.

10. Since there is no accepted "artificial mastoid" agreed upon by the Bureau of Standards or other major authority, some commercial bone conduction oscillators may not be the instruments of precision that they are reputed to be.

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247 Commonwealth Avenue.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

AND

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY JOINT MEETING.

Arrangements have been completed for a joint meeting of the South Carolina Society of Ophthalmology and Otolaryngology and the North Carolina Eye, Ear, Nose and Throat Society to be held at the Francis Marion Hotel, Charleston, S. Car., Sept. 14, 15 and 16, 1953. The following otolaryngologists will be on the program: Dr. Wayne Slaughter, of Chicago; Dr. John R. Lindsay, of Chicago; Dr. Theodore Walsh, of St. Louis.

A panel on vertigo will be discussed from the opththalmological, otolaryngological, neurological and internal medical standpoints.

Dr. Charles Kunkle, of Duke University, will present neurology, and Dr. Vince Mosely, of the Medical College of South Carolina, will be the guest internist.

The following three will cover the ophthalmological subjects: Dr. Derrick Vail, of Chicago; Dr. David G. Cogan, of Boston; Dr. R. Townley Paton, of New York.

FUNCTIONS OF THE NASAL SEPTUM AS RELATED TO SEPTAL RECONSTRUCTIVE SURGERY.*

RUSSELL I. WILLIAMS, M.D. (by invitation),
Cheyenne, Wyo.

When Freer,[1] of Chicago, published his classic summary of the submucous resection operation in 1902, he not only concluded the anatomical conception of this surgery but also suggested and indicated further investigations in pathology and physiology areas. He really brought to a focus 50 years of pioneering in this field of nasal surgery. During the following 50 years a few investigators emphasized the need for reconsideration of septal surgery; notably among these were a group of European surgeons and Metzenbaum[2] and Peer[3] in this country. During the last decade, because of a growing interest in surgery of the external nasal pyramid and its relation to the septum, the need for more extensive understanding of physiology of the nose has become most imperative. We know that the function of the nose is a much more significant and profound one than just warming, moistening and cleaning the air which we breathe. We are now concerned with the reasons for the superiority of nasal breathing over mouth breathing; the reason for having two nasal chambers; and the reasons for and the corrections of nasal insufficiencies. In this paper an effort will be made to take up a very small part of these problems, namely, the rôle of the septum, first as an independent single structure, and second as it functions in conjunction with its neighboring parts.

ANATOMY.

The usual anatomical presentation of the nasal septum includes a description of the perpendicular plate of the ethmoid,

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the vomer, the medial crests of palatine and maxillary bones, and the septal or quadrilateral cartilage. On either side of the septal framework is the mucous membrane, which becomes skin over the membranous septum and the columella. It consists of pseudostratified ciliated columnar epithelium, resting on the tunica propria, which is quite vascular, containing both serous and mucous glands and is adjacent to the perichondrium and periosteum. The mucous membrane is reflected from the septum to the under surfaces of the upper lateral cartilages, the nasal bones, and onto the lateral nasal walls. Superiorly, above the middle turbinate, the mucous membrane becomes olfactory, with a more columnar epithelium and without cilia. In the olfactory area, few glandular elements are noted in the tunica propria. The blood supply arises from the sphenopalatine artery and anastomoses freely with the ethmoidal, external nasal, and anterior palatine arteries. The veins of like names accompany the arteries basically draining into the sphenopalatine and ethmoidal veins. The nerve supply originates from the sphenopalatine ganglion, the nasal, ethmoidal and the olfactory nerves. The lymph drainage is into the superficial cervical lymph glands, the submaxillary glands, the retropharyngeal and deep cervical lymph glands.

SURGICAL ANATOMY.

Because septal surgery is no longer limited to the central portion of the septum only, but may also involve the whole structure to its peripheral limits, and because we must concern ourselves so frequently with the structures adjacent to the septum, a more meticulous inspection of septal construction is in order. On close investigation we see that the nasal bones contribute a perpendicular member to the formation of the septum. Beneath and supporting them, in the midline, is the nasal spine of the frontal bone, a structure of great importance. The anterior portion of the frontal spine is a part of the septum. Its continuity with the perpendicular plate of the ethmoid and the nasal bone is of particular concern in the mobilization of the septum and the external nasal pyramid. From a functional viewpoint, the frontal spine, the perpendicular plate of the ethmoid, the medial portions of the

nasal bones, the vomer, the medial crests of the maxillary and palatine bones, constitute the immobile portion of the septum. The semimobile portion is the septal or quadrilateral cartilage, which, together with the upper lateral cartilages,[4] constitutes the cartilaginous vault of the nose. The membranous septum is of great significance. It is folded over the caudal end of the septal cartilage so as to appear negligible, but is 8 to 10 mm. in length. Beyond this is the columella, containing the medial crura of the lobular or alar cartilages. It also subserves a septal or partitioning function. Beneath this in the midline extends the maxillary spine made up of projecting portions of both superior maxilla. This protects the incisive canals lying immediately posteriorly, which contain the nasal palatine nerves, after they run their courses over the septum on each side. The rostrum of the sphenoid also enters into the constitution of the septum at the posterior surface border. The total cartilaginous wall extends under the nasal bony arch for a variable distance and articulates with the perpendicular plate of the ethmoid in the midline in a firm union, which, however, does not adequately prevent the frequent detachment of the cartilage from the bone in this area following trauma.

The clinical abnormalities of the septum requiring reconstruction include dislocations, deviations, impactions, obstructions, spurs, and ridges. One of the most common is the anterior dislocation of the septal cartilage with a resulting deformity of the columella and distortion of the adjacent vestibular structures.

Most of this is common knowledge. Kreig's[5] pictures of the infinite variety of shapes that these deformities may assume are, of course, well known to all of you, but his pictures do not show all the essential pathology. The deformed cartilages may be fractured; there may be pieces completely broken off; some portions may be missing; scar tissue may invade the injured areas and replace and encapsulate the detached fragments. Scarring of the septum as the result of fractures may unite the mucosal flaps. Fragments of bone or cartilage deprived of their blood supply may become enclosed between

the mucosal flaps, with subsequent absorption of the bone and cartilage. Adhesions, from the one septal mucosal flap to the other and to the lateral nasal wall are a common occurrence.

A septum may be exceptionally long or it may be protruding anteriorly—increased projection. Portions of the septum may be absent as a result of infection or secondary atrophy, resulting in either case in a very thin septal mucosa or a perforated one.

Congenital malformations of the mucosa, the rostrum of the sphenoid, the palatine crests, etc., may occur.

A consideration of the anatomy of the septum would be incomplete without the consideration of its position in relation to the lateral walls of the nose. There is a great variation in the distances between these structures which may extend beyond the physiologically corrective capacity of the turbinates.

FUNCTION.

To say that the septum is the nasal partition tells nothing of its purpose. Why does it need to be hard and soft at the same time? Why is it fixed, semimobile and freely mobile in different areas? Why do we have a partition in the first place? A full discussion of these interesting topics will not be given, but a brief summary is herewith presented.

The partition creates two noses, each a distinct and complete entity. Each nose has a neurological connection with the basal nuclei in the medulla and thence with the nerve supply of the corresponding lung. It supports and helps create the nasal dorsum and makes in effect a roof for the nose—a tegmen. It provides a hard wall in the air passage which offers resistance to the air currents. It serves as the resisting terminal for the rhythmic excursions of the turbinates. It separates the medial crura. It supports the Schneiderian membrane. It supports the nasal bridge. In conjunction with the upper lateral cartilages, it completes the upper lateral valve[6,7] of the nose and in conjunction with the turbinates regulates the air flow and creates eddies of air currents. Together with the nasal bones, it helps to protect the cranial vault.

RECONSTRUCTION.

Since deformities of other parts of the nasal pyramid so often accompany a septum requiring reconstruction, it is obvious that an operative technique more extensive and fluid than the simple submucous resection is required; therefore, in reconstructions, our goal should be to retain or refashion a septum which has *1.* an immobile portion; *2.* a soft semi-mobile; *3.* a mobile portion; a septum which creates two noses; and one which lends support to the adjacent nasal structure. It should have an adequate relationship to the lateral nasal wall. An attempt should be made to prevent cicatricial contraction of the cartilaginous dorsum and the base of the nose, the columella. In addition, we should take steps to minimize the possibility of secondary atrophy and subsequent perforations. For this reason, injury to the mucosa, perichondrium and periosteum is to be avoided.

It is impractical to have a fixed method which is dogmatically followed. Each case will present its individual problems.

It is, therefore, necessary to acquaint ourselves with many different techniques and apply them where they would be most useful. There is no routine in this type of surgery. It is necessary to be plastic in our thinking in order that we may accomplish the desired result for the individual patient with the minimum of disturbance to the good portion or portions of a particular nose.

The surgical technique should allow good exposure of all parts of the septum and its adjacent structures. By means of the anterior septal incision[8] (Cottle incision) cephalic to the membranous septum, it is possible to expose the septum in its entirety and allow surgical procedures on all or any portion or portions of the septum. It is possible to remove, reset or replace the caudal end of the septum. The entire septal cartilage may be removed if necessary, or thinned sagitally; it may be gridded and molded; or it may be replaced by fresh or preserved human or ox cartilage. The bony portion may be split sagitally and thinned, or it may be moved, or removed,

and replaced. It is also possible to leave any good portion of the septum, either bone or cartilage, and remove or modify only those portions which are producing the disturbance.

Through this incision, surgery of the membranous septum may be performed easily: the columella lends itself well to modification. Abnormalities such as a hanging, wide, retracted, oblique, short, or long columella, or one with clefts, may be corrected.

Separation of the upper lateral cartilages from the septum intraseptally may be easily accomplished. It is equally feasible to separate the nasal bones intraseptally and to complete the work necessary to accomplish a good relationship of the nasal structures. The dorsum can be lowered. It can also be raised by use of implants of bone, cartilage, or plastics, without injury to the mucous membrane. Surgery of the nasal spine is relatively easy through this approach. Surgery of septal perforation is being developed.

Our present concept of reconstructive surgery of the nasal septum is to achieve as nearly normal anatomical and functional result as possible with the least amount of surgical trauma. In general, the best operation on the septum is the one in which the least amount of surgery is done. The anterior septal incision (Cottle incision) (hemitransfixion) makes it possible to obtain good exposure of all parts of the nasal septum, spine and dorsum and allow procedures on any portion of it with the least amount of trauma to the mucous membrane, the perichondrium and the periosteum. When we remove distorted or deformed portions of the septum and replace the septal cartilage and bone between the mucosal flaps we are making an attempt to restore normal anatomical relations of the septum.

The septum must be treated as an integral part of the nose. Surgery of only one portion without due consideration of the nose in its entirety will consistently result in a poorly functioning nose. Surgery of the septum must be combined with

an evaluation of its relationship to the lateral nasal walls, its relationship to the upper lateral cartilages, its relationship to the nasal bones, and its relationship to the lobule.

All of the above procedures may be carried out entirely from the anterior (Cottle) incision. It is possible to combine this incision with intercartilaginous and circumferential incisions to correct further deformities of the nasal pyramid and the lobule.

Corrective surgery of the nasal pyramid consists of: lateral osteotomies with infracture of the nasal bones, complete mobilization of the nasal pyramid, and lowering or levelling up of the dorsum. Very often corrective surgery of the cartilaginous vault and lobule should be combined with the septal operation.

Several common deformities warrant special mention. A flattened nose will remain a poorly functioning nose regardless of how well the septal surgery has been done. It is necessary to elevate the tip so that the air currents will be allowed to travel in the right direction. Large nostrils will not permit a good functioning nose unless proper relationship of the lower lateral cartilages and the upper lateral cartilages and the septum are restored. A crooked nose, a high narrow nose, a broad nose, a nose with child type nostrils and lobules in adults, probably the result of injury and subsequent retardation of growth, do not foster good nasal function regardless of how well septal surgery may be done. These disproportions must be corrected.

CONCLUSION.

Until recently the many procedures necessary to accomplish the desired end-results in septal surgery have not been too well developed and were considered difficult and dangerous; however, within the last decade numerous articles have been written on these subjects and facilities have been established for seeing this type of work performed, and for receiving class and personal types of instruction.

I am sure that we stand at the threshold of a new era in our specialty, and that all the experiences that have accumulated during the last hundred years constitute the background from which progress in rhinology and rhinologic surgery will develop.

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HEARING IMPAIRMENTS IN CHILDREN.*

CHARLES E. KINNEY, M.D.,
Cleveland, Ohio.

This paper is an attempt to analyze the hearing diagnosis made upon 3,622 children examined during the past 15 years in our Otological Diagnostic Clinic at Alexander Graham Bell School in Cleveland. The ages of these children varied from about two and one-half up to 17 years with the greatest concentration of first examinations being between the ages of eight and 11 years. These children were referred to this diagnostic clinic by technicians doing hearing screening tests throughout the city school system, by school physicians and by other community health agencies dealing with children.

The procedural methods in arriving at these diagnoses have remained about the same during these years. The otological histories were obtained, and the air conduction pure tone audiometry was done by a well trained nurse, Miss J. Arthur, for the first six years, and Mrs. M. Hlavin for the past nine years. I made the ear, nose and throat examinations as well as bone conduction audiometry and speech testing when necessary. In better than 95 per cent of these cases, one or both parents were present at the examination; therefore, it was possible to obtain very excellent histories, including the age, severity and complications of contagious diseases. The examinations were made in a room that had been acoustically treated, and the noise level ran between 50 and 55 db. It is obvious that in a fair percentage of these cases it was impossible to make a positive diagnosis until a therapeutic procedure had been attempted. These procedures were done by many different doctors; however, in about 85 per cent of such cases, we were able to get the child back for one or more fol-

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low-up examinations. In many of these cases it was obvious that there was a combined perception and conduction factor; however, every case has been classified as one or the other, depending upon which we considered the predominating factor.

In Table 1, one will see a breakdown of these cases into major groups. There were 367 cases classified as having no usable hearing. By this we mean that they had no better than

TABLE 1. ANALYSIS OF THE 3,622 CASES.

No usable hearing			367
Normal hearing			627
Conduction cases		1,688	
No hearing one ear, normal hearing other ear	310		
Partial perception cases	630		
Perception cases		940	
Cases further analyzed			2,628
Total			3,622

15 per cent hearing in their better ear. The fact that this group makes up 10 per cent of the entire series requires some explanation. Our metropolitan population is about 1,500,000 people. It is safe to say that during the past 15 years nearly every child in this area, that could be classified in this group, has been examined in our clinic. On the other hand, there were probably several hundred partial loss perception cases and several thousand conduction cases that we did not see in our clinic. These 367 cases, as well as the 627 cases in which the hearing was found to be normal in both ears, will not be further analyzed.

This leaves 2,628 cases to be studied. Of these, 1,688 (64.2 per cent) were classified as conduction cases and 940 (35.8 per cent) were classified as perception cases. About one-third (310) of the perception cases had completely normal hearing in one ear, with no hearing in the other ear. The other two-thirds (630) had partial impairment in one or both ears.

In Table 2, the diagnoses of the 1,688 conduction cases are analyzed. You will note that there are 298 cases of bilateral and unilateral active otitis media. This requires some explanation in view of the fact that in a previous paper I had reported that during the school year of 1935-1936 we had discovered 622 children with discharging ears. Those 622 children were discovered as a result of personal visits to the respective schools. At that time, as some of you will recall, no effective method of school hearing conservation had been established. It was not until the Fall of 1938 that the diagnostic clinic described in this paper was established. Between 1935 and 1938, the desirable results of public education as to the causes of otitis media and the increased use of the sulfa drugs had already proven itself. In this connection it was very interesting to have been sitting in my library about four weeks ago watching some television newscasts and to hear that a doctor in England had discovered that blowing one's nose was a cause of ear infections. I hope that you will pardon the personal reference.

TABLE 2. 1,688 CONDUCTION CASES.

	No. of Cases	% of These	% of Total
Active OM bilateral	156	9.2	5.9
Active OM unilateral	142	8.4	5.4
Healed OM bilateral	513	30.4	19.5
Healed OM unilateral	344	20.4	13.1
Tubal and/or adenoid	396	23.5	15.1
External otitis or wax	77	4.5	3.0
Middle ear effusion bilateral	17	1.0	0.7
Middle ear effusion unilateral	19	1.1	0.7
Otosclerosis	7	0.5	0.3
Congenital aplasia bilateral	4	0.3	0.2
Congenital aplasia unilateral	13	0.7	0.3
Totals	1,688	100.0	64.2

Although not shown on the chart, one can calculate that 1,155 (68.4 per cent) of these conduction cases have been due to otitis media, nearly every one of which could have been prevented. No comment is necessary about the adenoid cases

or the external ear cases. The middle ear effusion cases require some study. These 36 were all proven cases: they either had a very definite fluid level, or fluid was found when the middle ear was aspirated. I am sure that a fair number of the cases classified as healed otitis media were actually cases of middle ear effusion, but we were unable to prove this. These 36 cases have been diagnosed during the past four years. I feel that my interest in looking for these cases has increased during these years; further, I am quite convinced that there is both an actual as well as a relative increase in the number of these cases. Two of the seven cases classified as otosclerosis were girls who showed further loss in hearing after their first postmarital pregnancy. There were two sets of twins among these seven cases.

In Table 3, the 310 cases of unilateral hearing loss with normal hearing in the other ear have been classified. Of course, all of these are pure perception cases. When one of

TABLE 3. NO HEARING ONE EAR, NORMAL HEARING OTHER EAR.

	No. of Cases	% of These	% of Total
Unknown cause	131	42.3	5.0
Probable meningitis	74	23.9	2.8
Possible measles	72	23.2	2.7
Possible mumps	33	10.6	1.3
Totals	310	100.0	11.8

these cases is detected we try very hard to establish a cause by carefully questioning the parents and, in many instances, the child himself. We try to establish the timing and severity of contagious diseases with the discovery of the loss. In spite of these efforts, we were unable to establish a possible cause in nearly one-half of these cases. I have classified the meningitic cases as probable, because the history was rather definite. Incidentally, the incidence of these cases is on the upgrade because there are so many more of these children recovering from their meningitis. In trying to analyze the measles and mumps cases, I checked on the number of reported cases of these diseases in Cleveland proper (1,000,000

population) during the years 1937 to 1951. There were 66,348 cases of measles and 21,379 cases of mumps. This is a ratio of about three to one. Using this ratio, it would seem that mumps is more frequently the cause of unilateral total loss in hearing than measles. It is interesting to note that there was not even one hereditary case in this group.

In Table 4, the 630 partial perception cases have been analyzed. The word partial means that there was only a partial hearing loss. As mentioned previously, in the entire series of

TABLE 4. 630 PARTIAL LOSS PERCEPTION CASES.

	No. of Cases	% of These	% of Total
Unknown cause	273	43.3	10.4
Possible measles	109	17.3	4.1
Hereditary	92	14.6	3.5
Meningitis	30	4.8	1.1
Birth injury	24	3.8	0.9
Possible mumps	22	3.5	0.8
Cerebral palsy	19	3.0	0.7
Possible pertussis	17	2.7	0.6
Mother ill at 8 weeks	15	2.4	0.6
Erythroblastosis	14	2.2	0.5
Possible polio	6	1.0	0.3
Fractured skull	5	0.8	0.3
Mother, quinine 8 weeks	4	0.6	0.2
Totals	630	100.0	24.0

cases there has been no attempt made to classify cases as having a combined perception and conduction factor in their cause. Approximately one-half of these partial loss cases were unilateral. It was thought that the breakdown of this point by each etiological agent would not be of importance. Here again you will notice that in spite of extensive history questioning, we were unable to establish a cause in 43.3 per cent of the cases. You will notice that in these partial loss cases the ratio between measles and mumps is five to one. This is much higher than the ratio incidence of these two diseases; therefore, it would seem that measles may play a larger part in producing partial hearing loss than had been thought. You will also notice that the hereditary factor plays a large part

in partial loss cases as well as total loss cases. As I have advocated in several previous publications, this is an etiological factor that could be outbred among humans if we would only take the time to make it known by public education. The difference between the birth injury cases and the cerebral palsy cases should be explained. In the former, there was a definite history of long and complicated labor, and there were no other symptoms than the hearing loss; while in the cerebral palsy cases, there was not a single instance in which there was a difficult delivery history, but there were other symptoms in addition to the hearing loss. In the last line, you will note that there are four cases in which the mother took large doses of quinine at about the eighth week of pregnancy in order to effect an abortion. We have five cases in our total bilateral loss group that have been classified in a similar manner.

In Table 5, there is shown the analysis of hearing tests in the 630 partial loss perception cases. There were 461 cases that had had more than one hearing test. The average num-

TABLE 5. ANALYSIS OF HEARING TESTS, PARTIAL LOSS PERCEPTION CASES.

Cases tested average 2.9 times	461
Cases tested once only	169
Total	630

REPEAT TESTS.

No change	445	96.5 per cent
Progressive loss	16	3.5 per cent

ber of tests per child among these 461 cases was 2.9 tests. There were 169 cases that had only one examination. Of these 461 cases that had had one or more repeat examinations, 445, or 96.5 per cent, showed no appreciable change in their hearing. The average time elapse between the first and last examination of these 461 cases was four and one-half years. There were 16 cases (3.5 per cent) which showed a progressive loss of hearing. A very detailed study of these 16 cases is to

be presented in a paper later this year. At this time I can say that the progressive loss was definite and to an extensive degree. No true cause can be established, but I am prepared to state that the progress was not noted until a wearable hearing aid had been put on these children; in every instance the hearing loss was more marked in the ear in which the hearing aid was used. If we are prepared to accept the fact that sustained sound levels of high intensity can cause permanent cochlear damage in an otherwise normal ear, it is not too much to accept that this phenomenon may prevail in a previously damaged cochlea.

SUMMARY.

1. The causes of hearing loss in 2,628 children has been analyzed.
2. The ratio of conduction loss cases to perception loss cases is two to one.
3. A large percentage of the conduction loss cases was due to otitis media, nearly all of which could have been prevented.
4. Middle ear effusion as a cause of hearing loss in children is on the increase.
5. The measles virus as a cause of perception hearing loss in children is probably of greater import than has been reported in previous publications.
6. Although the frequency in which we encounter progressive perception hearing loss in children is not very great, when it occurs it is of serious consequence and it may be as a result of some of our modern therapeutic endeavors.

10515 Carnegie Avenue.

CYSTS IN THE VENTRICULAR AREA OF THE LARYNX.*†

HERMAN I. LAFF, M.D.,
Denver, Colo.

Cysts in the ventricular area of the larynx deserve further study and emphasis, and many facts regarding pathogenesis and clinical significance of the congenital variety need clarification. They do not resemble the more common laryngeal cysts sufficiently to be classified with them. Some cystadenomas, carcinomas, prolapses and particularly laryngoceles have many characteristics in common with ventricular cysts, which make their differentiation important.

The common type of laryngeal cyst represented by that most frequently found near the epiglottis need be mentioned only parenthetically. It is a retention cyst of an obstructed mucus gland. Similarly, another group on the vocal cords, called hemorrhagic cysts, are not true cysts, being lined only by a connective tissue capsule. Pathologically, in a true cyst the cavity should be lined with a secreting epithelium.[1] Lymph, traumatic and implantation cysts of the larynx which have been described are really only pseudocysts.

The terms congenital or embryonic cysts have been used to identify those rare cases found in the ventricular region of the larynx in the newborn. They also have been applied to similar cysts in adults.

The majority of congenital cysts in the newborn have been found at postmortem.[2] We should stress the importance of being more alert to their possible existence in the living infant. I have not encountered such a case to my knowledge.

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†From the Department of Otolaryngology, University of Colorado Medical Center, Denver, Colo.

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Pathologists whom I have interviewed also have affirmed a lack of experience with this entity, although some have intimated that these could be overlooked at postmortem since the larynx is not routinely removed in infants. Davidson,[3] in 1943, stated that only 15 cases had been reported to that date, the majority found at autopsy. Holinger and Steinmann,[4] in 1947, described two cases in infants who recovered. When discovered by direct laryngoscopy in dyspneic, cyanotic infants, rupture of the cyst brought dramatic relief.

Various opinions have been expressed regarding the origin of these cysts. A few authors have found them to be due to aberrant thyroid cells, others to displaced cells of branchiogenic origin. The majority have believed they were the result of developmental error arising in the ventricle or its saccule, such as the pinching off of the latter by displaced cells. Davidson[3] thought they represented a congenital anomaly associated with the early embryologic development of the larynx, similar to that found in atresia of the esophagus and duodenum. The vesicle representing the ventricle in the three-month-old fetus is connected with the lumen of the larynx by a solid epithelial cord of cells. Failure of this solid cord to become hollowed out may result in the formation of a cyst lined with columnar epithelium.

It also may be postulated that this could explain the origin of those cysts which only become manifest in adult life. The anlage may remain dormant and symptomless until trauma, infection or some other stimulus excites added growth.

I have treated three adult patients with cysts in the ventricular area of the larynx, forming the basis of this report. Because of their rarity, those previously recorded usually have been isolated cases. The variety of opinions still held regarding their origin and clinical significance renders their further study intriguing.

The first description of congenital laryngeal cyst was made by Abercrombie, in 1881. Louys has been credited with being the first to remove one by throtomy.

More recently, Davis,[5] in 1923, described three cysts of the neck which penetrated the thyrohyoid membrane and extended into the wall of the larynx.

Eggston and Wolff[6] quoted Wassler as having described a laryngeal cyst which he considered to have originated from embryonic displaced thyroid tissue.

Imperatori,[7] in 1929, reported a multilocular branchial cleft cyst of the larynx, removed by thyrotomy.

New and Erich,[8] in 1939, stressed the extreme rarity of congenital cysts of the larynx, reporting only one case in 722 benign laryngeal tumors, among which were 35 cysts. They added two cases, one of which they found while performing laryngectomy for an extensive epithelioma. They discussed the branchial cyst origin of these tumors, pointing to the fact that the larynx is formed from the fourth and fifth branchial arches. A branchial cyst, they stated, could occur as readily mesial to the thyroid cartilage as external to it. Holinger and Steinmann,[4] however, after comparing the embryology of the larynx and branchial arches, contended that tumors caused by displaced cells from the latter would have to lie outside the thyroid cartilage and never inside the larynx, as do congenital cysts. New and Erich[8] stated: "Microscopically, both types of cysts bear some points of resemblance, since both may be lined with stratified columnar epithelium; however, in the walls of the branchial cysts are aggregations of lymphoid tissue that contain germinal centers (see Fig. 1). The finding of lymphocytic infiltration alone is generally regarded insufficient to designate the cyst branchiogenic (see Figs. 3 and 4). They concluded that congenital cysts probably originate as a result of sequestration of embryonic cells from the appendix of the ventricle.

Leonardelli and Nicelli,[9] in 1949, expressed the view that a branchial cyst may press inwards and invade the pharynx and larynx. This opinion appears not to conflict with stated embryologic facts and could explain the few branchial cysts discovered in the larynx.

El Hakeem,[10] in 1951, described a trilobed congenital laryngeal cyst having both internal and external extensions. It was lined with ciliated epithelium. Trauma appeared to have stimulated its development.

Clinical signs and symptoms of ventricular area cysts will obviously depend upon their size and whether they are only intrinsic in the larynx or connected with an extrinsic portion.

The patient will usually come in complaining of either hoarseness or dyspnea; cough, a desire to clear the throat, and a sense of fullness may also be noted. These symptoms, being present in many laryngeal states, will not be conclusive.

In the mirror a smooth, rounded, bulging mass will be visualized occupying the region of the false cord alone or including the adjacent part of the epiglottis, pyriform sinus, and aryepiglottic fold. Depending upon the size of the cyst, all or a portion of the vocal cord on the same side may be obscured. Its wall will have a smooth, shiny appearance with blood vessels coursing over it. It may be compressible by a probe. When the cyst has expanded into the neck above the thyroid cartilage, the soft, painless swelling may be palpated, pulsation will be absent, and the skin over it freely movable. It cannot be emptied by pressure, and cough will have but slight effect on its size.

The exact diagnosis of a smooth swelling in the ventricular area of the larynx may not be obvious, especially if it be small and deep.

I have been impressed, during routine mirror examinations of the larynx, by the occasional finding of assymetry of the two ventricular bands in patients who often had few laryngeal complaints. This was thought to be present when the vocal cord on one side was observed to be partly obscured by an enlarged, overhanging false cord. Such abnormality may represent only simple hyperplasia of the muscle, glandular and other contents of the false cords, but the thought of overlooking an early tumor or cyst has always been disturbing. When I performed biopsy on some of these the report generally stated that only excessive glandular elements were found.

Adenoma and cystadenoma originating in the glands of this region, as was pointed out by Figi, Rowland and New,[11] in 1944, is rare but of considerable interest. Up to that time 20 cases had been reported. They added four cases, three removed by endolaryngeal methods and one by thyrotomy. They stressed that its exact nature can be determined only by histological examination, as its gross appearance and the character of its fluid contents are not diagnostic.

Prolapse of the ventricle of Morgagni has been confused with cystadenoma. Koschier[12] has described two such cases.

The laryngologist's responsibility to bring suspicious and malignant growths of the larynx to early operation has been a chief factor in establishing the correct diagnosis of cyst. This has been the case in the three patients herein reported. The preoperative diagnosis in two was carcinoma and in the other, cystadenoma.

When carcinoma is primary in the mucosa of the ventricle the direction of growth in some may be toward the laryngeal lumen, giving the appearance of a prolapse; in others, the growth will extend upward and outwards, pressing the false cord and aryepiglottic fold in front of it. The resulting swelling may be mistaken for a cyst until ulceration, which may be long delayed, becomes evident. Sarcoma, likewise, may produce a diffuse infiltration of the ventricular band.[13]

The presence of an external extension of the cyst may add to confusion if this be mistaken for glandular metastasis (Case 2). Movement of the cystic extension with swallowing and its compressibility should help to differentiate it from malignant lymph nodes. On the day prior to examination of the second case I saw a patient with unmistakable ventricular carcinoma, complicated by cervical metastasis, who presented an almost identical picture.

In my opinion some ventricular cysts originated as air sacs in which the air later became replaced by fluid. These air sacs have been called acquired laryngoceles to differentiate them from the true congenital variety associated with a congeni-

tally abnormal development of the saccule. Acquired laryngoceles may form in individuals in whom there is present interference with the normal exit of air from the ventricle.[14,15] This may result when inflammatory changes at the mouth of the ventricle or its saccule have produced a valvular type of obstruction, allowing air to get in but not out.[16] When inflammation has produced complete stenosis, the air becomes absorbed, allowing fluid to accumulate, thereby converting what previously has been a laryngocele into a cyst. This mechanism may be likened to that causing mucocele of the frontal sinus. Thomas[17] may have had this in mind when he described four cases of "saccular distention cysts." Similarly, Meda[18] recently called attention to the association of laryngoceles with cancer of the larynx. He was able to demonstrate them by Roentgenological examination, attributing their development to the same check valve mechanism.

Holinger and Steinmann[4] also stressed the identical location of laryngoceles and congenital cysts. In their opinion the two cannot be dissociated genetically. They look upon congenital cysts in infants as due to disturbances in development of the larynx and view such formations in adults probably as being "secondary isolated laryngoceles."

The type of epithelium found in the walls of the cysts affords no true diagnostic differentiation as both ciliated columnar and stratified epithelial lining have been noted in them as well as in laryngoceles. In some cysts prolonged pressure of the fluid produces atrophy of the lining. Richards[19] recently described squamous epithelial lining in the external portion of a laryngocele and respiratory lining in its internal extension. The laryngeal saccule normally is lined with a mucous membrane covered with columnar epithelium. Beneath the mucosa there is found adenoid tissue and lymphatic elements which have led to the designation of "laryngeal tonsil." Similar aggregations of lymphocytes, devoid of germinal cents, also have been found in the walls of congenital cysts. Their fluid content has not yielded characteristics of diagnostic value.

Although final proof admittedly is lacking, it is difficult to ignore the striking points of similarity between many ventricular area cysts and laryngoceles. The two certainly should be placed side by side in future discussions.

Regarding treatment, large ventricular cysts should be operated when diagnosed, since rapid increase in their size, as by infection, might dangerously encroach upon the airway. Preliminary evacuation of their contents may sometimes be desirable. In most cases, because of slow development, dyspnea surprisingly may be absent. Tracheotomy should precede operation on the larger cysts.

In infants, the use of forceps to open and evacuate the cyst will bring dramatic relief.

The majority of ventricular cysts in adults remains confined to the interior of the larynx. Although New and Erich[8] were able to remove some small ones by use of suspension laryngoscopy, recourse to thyrotomy generally will prove more satisfactory. Obtaining adequate tissue for biopsy may be difficult because of the elastic character of the cyst wall. When the cyst has extended outside the larynx it will be necessary to combine thyrotomy with an infrahyoid approach to dissect the portion of the cyst in the neck. I was able to remove intact both lobes of one such cyst. In another case I perforated the cyst wall, allowing a little fluid to spill. Air also was heard escaping, judging by the hissing sound. The possible "laryngocele" nature of this cyst obviously came to mind. The third case was treated by another laryngologist who performed a tracheotomy, followed by direct laryngoscopy, and succeeded only in partially removing the cyst wall. It is planned to extirpate the entire cyst by thyrotomy as soon as the patient gives her consent.

The total surgical removal by thyrotomy of the cyst confined to the interior of the larynx should offer no unusual difficulties. Its complete enucleation when it has an extension in the neck will be facilitated by adding a horizontal incision to the upper extremity of the vertical incison employed in thyrotomy. As El Hakeem[10] has pointed out, there are three

places where difficulty may be experienced in dissection: *1.* where the intermediate tendon of the digastric muscle may be adherent to the uppermost part of the cyst; *2.* near the upper margin of the thyroid cartilage where the hourglass constriction between the outer and inner compartments usually occurs; *3.* at the deep surface of the cyst where it leaves the thyrohyoid membrane, the superior laryngeal nerves and vessels having become adherent to the cyst wall.

Cystadenomas located in the false cord also are most suitable for enucleation by thyrotomy.

CASE REPORTS.

Case 1: H. K., white male, age 60, was seen in consultation on Feb. 2, 1950, because of a mass in the left side of the larynx. He has had Buerger's disease for many years, as a result of which he had one leg amputated 20 years ago and the other, four years ago. He has noticed intermittent hoarseness for over a year. His physician discovered a rounded swelling, about 2 x 3 cm. in diameter, near the angle of the left jaw and suspected malignancy. He was referred to another physicion, who, in addition, found a smooth, bulging swelling in the left half of the larynx largely obscuring the left cord. There were no palpable glands in the neck, nor was the thyroid gland enlarged. Carcinoma of the larynx "in-

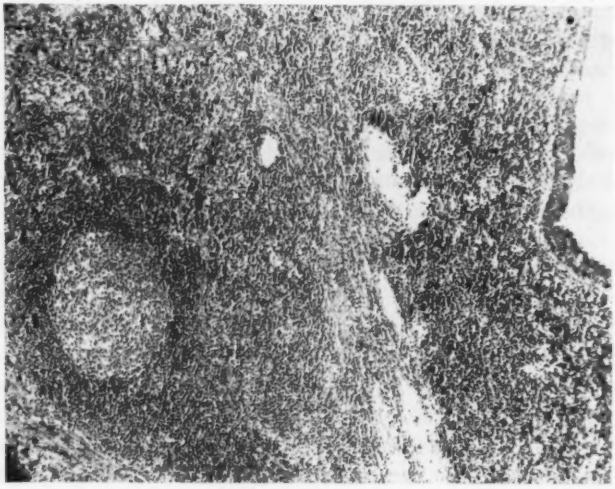

Fig. 1. Section to show typical findings in the wall of a branchial cyst. Note the stratified columnar epithelial lining and aggregations of lymphoid tissue containing germinal centers.

vading the musculature" was suspected. Direct laryngoscopy confirmed the absence of ulceration on the surface of the tumor. It appeared to reduce the patient's airway by about 50 per cent.

I performed a second direct laryngoscopic examination and found the laryngeal tumor to have a cystic consistency. Fragments removed for biopsy showed only normal laryngeal mucosa. My preoperative impression was cystadenoma. The mass which was excised from the left submandibular area showed findings typical of thromboangiitis obliterans.

On Feb. 13, 1950, I removed the cystic mass by laryngofissure, preceded by tracheotomy. It lay in the left side of the larynx above the left cord, from the base of the epiglottis to the arytenoid and extending upward into the aryepiglottic fold. While freeing the cyst, a sound simulating the escape of air was noted on two occasions and the possibility of the existence of a laryngocele came to mind. The cyst was inadvertently nicked late in the course of its dissection when a small amount of grayish gelatinous material also spilled.

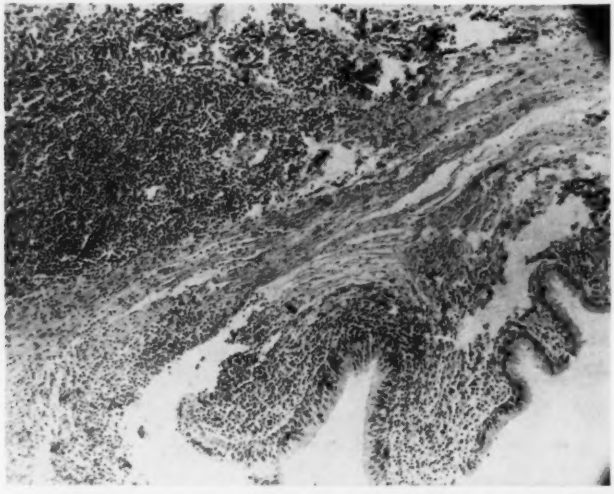

Fig. 2. Section through wall of Case 1. Note the columnar epithelial lining, the lymphocytic infiltration and absence of distinct lymph follicles.

The pathologist's report follows: "Tissue specimen consists of an oval-shaped pouch which measures 2.5 x 4.5 cm. (see Fig. 4). The wall was broken before the specimen was submitted. The wall of the sac is 1 mm. or less in thickness, with several nodular areas of thickening upon the inner surfaces which appear somewhat rough and hemorrhagic.

"Microscopic examination shows the wall made up of rather heterogeneous elements. The surface shows stratified, squamous epithelial integument whose baseline is sharply defined, clearly outlined. In the

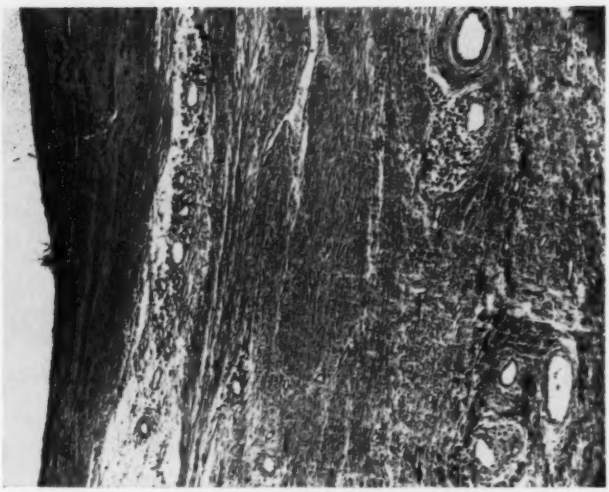

Fig. 3. Section through wall of Case 2. Note absence of epithelial lining which has disappeared as the result of atrophy.

fibrous tissue matrix there are large hypertrophic clusters of mucus glands and ducts. The inner surfaces show small patches of epithelial lining and plasmocytic infiltration. Small areas of hemorrhages and fibroblastic cell proliferation are seen. No evidences of neoplastic changes are seen at any point, and all cellular components appear to be well differentiated (see Fig. 2). *Diagnosis:* Cyst of the laryngeal mucosa, with secondary chronic inflammatory changes."

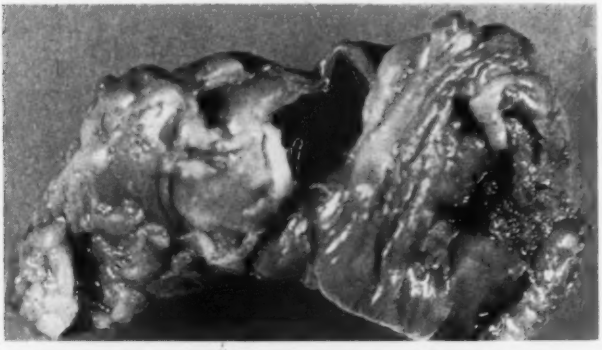

Fig. 4. Cyst removed from Case 1. It measured 4.5 cm. x 2.5 cm.

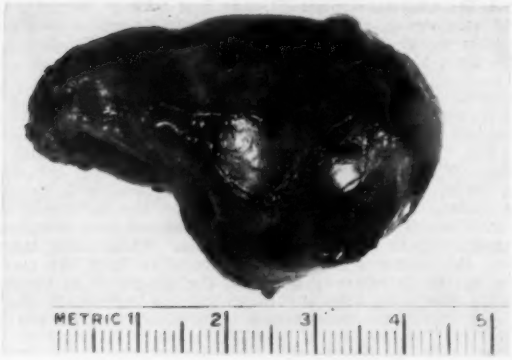

Fig. 5. Cyst removed from Case 2.

The patient's postoperative course was uninterrupted. He was discharged from the hospital on Feb. 20, 1950. There has been no evidence of recurrence to date.

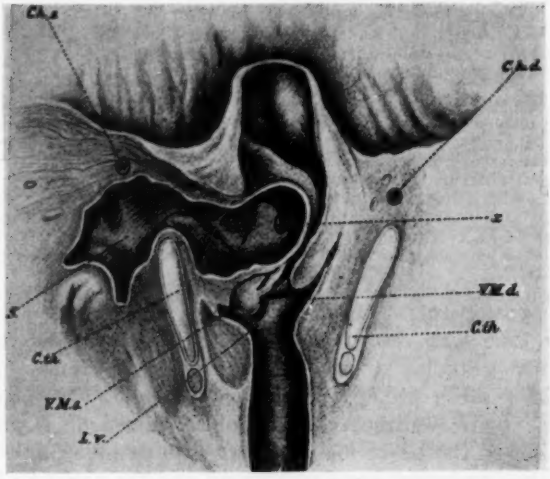

Fig. 6. Drawing to show relation of laryngocele and congenital laryngeal cyst to structures of larynx and side of neck. The relations of each are the same. (After Hajek.)

Case 2: A. H., white male, age 57, was first seen on Nov. 26, 1951, complaining of hoarseness, slight dyspnea, dysphagia and a swelling of the left side of the neck. The hoarseness came on gradually during the past six months. Past history was irrelevant.

Mirror examination showed a rounded, smooth mass in the left side of the larynx involving the false cord and aryepiglottic fold, extending past the midline and encroaching into the base of the epiglottis, vallecula and pyriform sinus. The left true cord was completely covered and could not be visualized. There was a swelling in the left side of the neck below the hyoid bone, about 2 cm. in diameter, presumed to consist of metastatic nodes. During direct laryngoscopy the laryngeal mass was found to have a cystic feel. The tissue obtained was reported normal laryngeal mucosa with glandular hyperplasia. When, after more careful examination, the external swelling was found to have the same elastic consistency as the intralaryngeal mass, the diagnosis of laryngeal cyst with external extension into the neck was made. Air in the sac was excluded by inability to demonstrate it by X-ray or to force it out by pressure.

Operation was performed on Jan. 3, 1952, preceded by tracheotomy. A midline incision was made from the hyoid to the jugulum with a horizontal arm extending from its upper end to the left sternocleidomastoid muscle. After the skin flaps were raised, midline thyrotomy was performed. The infrahyoid muscles on the left side were severed from the hyoid, which was then split in its midportion, facilitating dissection. The mucosa and deeper structures covering the laryngeal portion of the cyst were then separated and the entire cyst removed complete.

The pathological report follows "Specimen consists of a roughly pear-shaped, 4.5 x 3 x 2 cm., cyst (see Fig. 5), with a shallow indentation separating the wider from the narrower portion. The cyst has a thin, translucent, yellow-gray wall and in some places is covered with small patches of fibrous tissue. It contains gray, turbid mucoid material. The inner surface is light gray and smooth; the wall is up to 1.5 mm. thick.

"Microscopic examination (see Fig. 3) shows the inner surface of the cyst is devoid of epithelial lining for the most part; however, inconspicuous small sheets of flattened or low-cuboidal cells with dark round or oval nuclei are present in some places. The wall of the cyst is made up of long, straight or curved bundles of moderately vascular connective tissue with a small number of fibrocytes. Circumscribed infiltrates of lymphocytes and plasma cells are present in a few places close to the inner surface. Small clusters of seromucinous glands are found occasionally in the outer layers or detached from the wall. *Diagnosis:* Cyst, laryngeal, congenital."

The postoperative course was uneventful and the tracheotomy tube was removed on the fourth day. Two months later, a granulomatous mass protruding from the left ventricle, like a prolapse, was removed by forceps and cauterized during indirect laryngoscopy. Microscopic examination showed the tissue to be composed of granulations. Since then, the "prolapse" has disappeared and the patient's larynx and voice are nearly normal.

Case 3: N. P., white female, age 60, entered Colorado General Hospital on Sept. 7, 1948, with the complaints of hoarseness, difficulty in swallowing and dyspnea. Laryngeal examination revealed a large, irregular mass in the substance of the left ventricular band extending from the anterior commissure to the left arytenoid. No masses were palpable in the neck. She had been referred by her local physician for treatment of possible cancer of the larynx. Because the tumor was obstructing the airway, tracheotomy and biopsy were performed Sept. 9, 1948. About one-half teaspoonful of purulent-like material was released from the mass when the biopsy was taken, the postoperative clinical diagnosis being "probable benign cyst of larynx."

The pathologic report follows: "A translucent, white, gel-like ball measuring 5 mm. in diameter and two fragments of firm, torn tissue were removed.

"Microscopic examination reveals a mass of amorphous polychromatic material in which a few partially disintegrated leucocytes and squamous epithelial cells are identified. It appears to be the extruded contents of a cyst. In addition are fragments of laryngeal wall and vocal cord, covered by regular, pseudostratified, ciliated columnar epithelium and stratified squamous epithelium, respectively. Identified are normal groups of mixed glands. *Diagnosis:* Laryngeal tissue, no diagnostic features."

When last seen in June, 1952, the mass in the left ventricular area was still present. Thyrotomy has been recommended.

SUMMARY AND CONCLUSIONS.

A brief survey of cystic swellings in the ventricular area of the larynx has been presented in order to emphasize their etiological and clinical importance.

The congenital variety, as found in infants, most likely is due to maldevelopment of the ventricle or its appendage, the saccule. Early diagnosis and treatment are stressed.

Some congenital cysts in adults have a similar origin; others probably commence as laryngoceles which later convert into cysts.

Laryngoceles and congenital cysts are closely allied genetically and should be considered as manifestations of the same developmental error.

Embryologically, branchial cysts probably are incapable of developing in the larynx. They may invade the larynx by extension from their usual site of origin in the neck.

Other cysts and tumors found in the ventricular area of the larynx also are discussed.

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227 16th Street.

SPONTANEOUS CEREBROSPINAL RHINORRHEA SECONDARY TO THE ARNOLD-CHIARI MALFORMATION.*

NELSON A. YOUNGS, M.D.,‡
Grand Forks, N. Dak.,
and
WILLIAM PEYTON, M.D.† (by invitation),
Minneapolis, Minn.

The Arnold-Chiari malformation is a downward displacement or herniation of the cerebellum and brain stem through the foramen magnum into the upper part of the vertebral canal. This condition was first described in 1894 by Arnold and in 1895 by Chiari.

In addition to the herniation of parts of the hind brain through the foramen magnum, the cranial nerves arising from the pons and medulla are greatly elongated and pass upward to reach their foramina of exit from the skull, also the roots of the upper cervical segments ascend to their exit through the cervical intervertebral foramina.

There is still much disagreement on the pathogenesis of this deformity and little concurrence regarding its relationship with platybasis and spina bifida. Penfield and Coburn[13] considered the Arnold-Chiari malformation to be brought about by traction on the brain stem during embryonic life, resulting from fixation of the spinal cord at the site of a meningocele. Normally in intrauterine life, growth of the vertebral column and spinal cord occurs at an equal rate until the third month. After this, growth of the bony vertebral

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†Department of Neurosurgery, University of Minnesota.

‡Department of Otolaryngology, Grand Forks Clinic, Grand Forks, N. Dak.

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canal outstrips the cord, and the spinal cord is normally pulled upward so that the conus medullaris eventually in adult man comes to be opposite the first lumbar vertebra.

In myelomeningocele, the cord becomes fixed at the site of the sac so that traction is exerted on the cord and that part of the brain stem below the tentorial notch by the continued

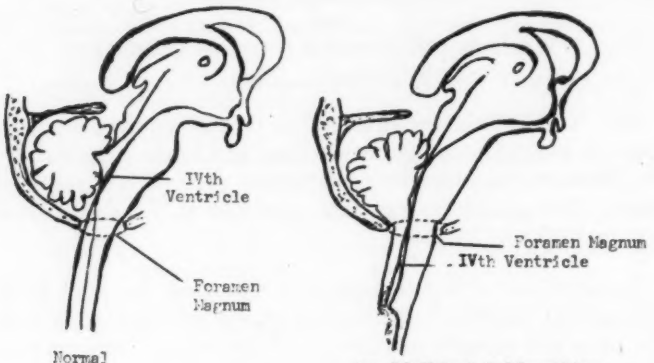

Fig. 1. Comparing Arnold-Chiari Malformation with Normal.

growth of the vertebral column. Thus a portion of the hind brain is drawn through the foramen magnum, producing this deformity.

In 1938, Aring[2] reported a case of Arnold-Chiari malformation in an adult with cerebral symptoms without associated hydrocephalus or spina bifida. McConnell and Parker, in 1933, reported five cases apparently not associated with spina bifida, occult or cystic. Adams, Schatzki and Scoville,[1] in 1941, reported two cases, one associated with myelomeningocele and the other not. Ogryzlo[12] reported seven cases in 1942, three without apparent spina bifida, three with associated myelomeningocele and one with simple meningocele. Bucy and Lichtenstein,[3] in 1945, reported a 40-year-old female with the deformity without spina bifida. They commented, "the occur-

rence of an Arnold-Chiari deformity of sufficient magnitude to produce marked neurologic symptoms with no evidence of a spina bifida or any bony anomaly in the cervical region is most unusual."

Hydrocephalus, although usually present, is not an essential feature of this condition. According to Donald and Russel[15] hydrocephalus is brought about in three ways:

1. An obstructive type. In this type the foramina of Luschka and Magendie are obliterated by compaction of the hind brain into the foramen magnum.

2. A communicating type. In this type the fourth ventricle is below the foramen magnum with the foramina of Luschka and Magendie patent so that the cerebrospinal fluid from the ventricles is able to flow into the spinal subarachnoid space, but constriction at the foramen magnum causes a block to exist between the spinal and cerebral subarachnoid spaces, thus preventing the reflux of this fluid back into the cranial cavity to the cerebral subarachnoid spaces where most cerebrospinal fluid is normally absorbed.

3. An adhesive type. In this type of hydrocephalus, traction on the brain stem causes a mechanical irritation which in turn causes adhesions to occur in the region of the foramen magnum, thus blocking the flow of cerebrospinal fluid from the spinal subarachnoid space into the cerebral subarachnoid spaces.

My reasons for calling your attention to this condition by this brief review of the literature and the report of a case are, first, because this lesion apparently far removed from otolaryngology may give its first or dominant symptoms in our field; second, I wish to record a case of Arnold-Chiari deformity in an adult with a presenting symptom of cerebrospinal rhinorrhea, a presenting symptom which I have not found previously reported.

Ogryzlo[12] lists the following signs and symptoms as those most usually found in this deformity:

1. Early morning headaches
2. Ataxia and staggering gait.
3. Nystagmus.
4. Palsies of the cranial nerves.
5. Visual disturbances.
6. Papilloedema.
7. Vomiting.
8. Pain in the occipital, suboccipital and cervical regions, sometimes relieved by stretching or forward flexion of the neck.
9. Paresthesis of the upper extremities.
10. Symptoms aggravated by coughing, sneezing, and straining.

REPORT OF CASE.

Mrs. W. K., white female, age 29, first seen Sept. 6, 1950, complaining of copious amounts of watery nasal discharge for the past six months. Our examination revealed a normal ear, nose and throat. It was our thought that we might be dealing with an allergic rhinitis during a quiescent period. The patient was placed on an antihistaminic and asked to return if her symptoms persisted.

The patient did not return until Feb. 22, 1951, when she stated her symptoms were the same. Further direct questioning brought out the fact that this watery nasal discharge came only from her left nostril and was most severe upon arising in the morning or whenever she, as a stenographer, leaned over her desk.

Examination at that time revealed the pupils to be equal and reacted to light and accommodation. There was a slight but definite nystagmus on looking towards the left with a vertical component. The skin around the left nostril appeared to be irritated, but there was no fluid in abnormal quantities in either nostril and each nasal cavity appeared normal. The septum, turbinates and the nasopharynx were all normal to inspection. The tonsils were out, the teeth appeared normal, and the sinuses transilluminated well. The functional hearing tests, including an audiogram, were all normal. The response to the cold caloric test was normal in each ear.

Neurological examination was negative except for minor changes, but the consultant did suggest this as a cerebrospinal fluid fistula, because when the patient sat on the examining table a clear, watery liquid gushed out of her left nostril in quantity. When asked to collect some of this fluid, she did so by alternately coming into the upright from the

supine position. Each time she sat up, from 1 to 2 cc. of clear fluid came from the left nostril. The fluid thus collected was clear and contained six lymphocytes, 3 pmn. per cu. mm. and had a sugar content of 75 mgm. per cent.

X-ray examination of the skull revealed evidence of increased intracranial pressure, with increase in gyral markings and some enlargement of the pituitary fossae.

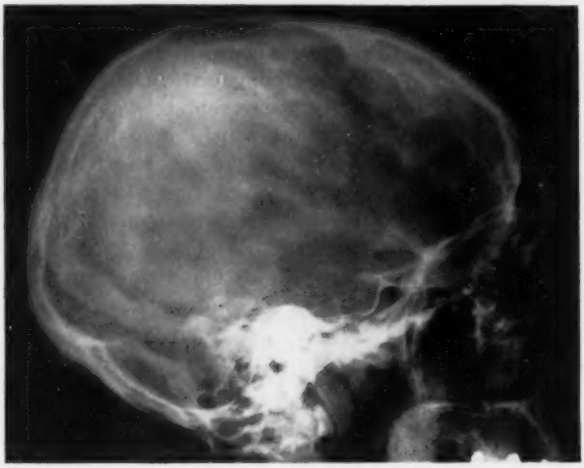

Fig. 2. X-ray of the skull showing the sella to be a little enlarged. Digital markings increased. Dorsum snubbed down. No decalcification of floor.

This patient then was referred for neurosurgery (W. T. P.). Neurological examination again revealed minimal signs of a cerebellar lesion. Because of this and because the Roentgenograms of the skull showed evidence of chronic increased intracranial pressure, a carotid angiogram was done on the right side. This showed evidence of a hydrocephalus. Some methylene blue was inserted into a lateral ventricle and none of it was recovered on later spinal puncture. A ventriculogram was made, and this showed a very large bilateral hydrocephalus. It was, therefore, concluded that the probable cause of the rhinorrhea was increased intracranial pressure and that the increased intracranial pressure was due to some benign stenosis of the aqueduct. It was decided that a Torkildsen procedure (tube drainage from lateral ventricle to cisterna magna) should be done. Under intratracheal anesthesia in the sitting posture, the posterior fossa was exposed by a midline incision. There was enormous dilated venous channels in the occipital bone that bled most vigorously until controlled, unusual difficulty being encountered because of their size. These enlarged anastomotic veins are due to prolonged compression of the intracranial veins and sinuses. When the dura and the posterior fossae were opened, adhesions were found around the foramen magnum, especially in the cisterna magna. The tonsils were found to extend through the foramen magnum so that the first, second and third

cervical vertebral lamina had to be removed to get to the lower end of the tonsils projecting down into the cervical canal. They extended down as far as the lower edge of the third cervical lamina and completely filled the spinal canal. When the cerebellar tonsils were thus freed, the blue dye which had been injected into the lateral ventricle first appeared and came freely into the wound.

SUBSEQUENT COURSE.

The immediate postoperative course was uneventful. Her rhinorrhea had practically disappeared by the time she was discharged from the hospital 13 days after the operation, and she continued to be entirely free from this symptom for a period of about four months, at which time she again noted two or three drops of clear fluid from her left nostril each morning on arising. At the present time, 12 months after operation, she has about seven or eight drops of clear fluid from this left nostril on arising, but at no other time during the day. It is apparent that this fistula, through which cerebrospinal fluid is escaping into the nose, still must be closed surgically.

Another interesting phase in this woman's recovery was the improved pituitary function. She had had a persistent amenorrhea for nine years, or since she was 19 years of age. Although married and desirous of having children, yet she had never become pregnant until after surgery, when menses reappeared and after three normal periods she became pregnant. She delivered a normal male infant on March 2, 1952, one year after operation. Obviously, pituitary function was impaired by pressure of the floor of the hydrocephalic third ventricle.

A short time after surgery for the Arnold-Chiari malformation, the patient developed signs and symptoms of a herniated disc, which again necessitated surgery. In the course of studies of the patient's spine, preliminary to surgery for this disc lesion, a spina bifida occulta of a lumbarized first sacral vertebra was noted. I have noted this because, although some authors have reported simple meningocele associated with and apparently causing Arnold-Chiari malformation, I have been unable to find any previous report of a relationship between spina bifida occulta and this deformity.[1-8,11,14,16]

Following surgery for the herniated disc, July 9, 1952, the patient has been able to carry on as a normal housewife, taking care of her infant son and husband, but she continues to have seven or eight drops of clear fluid from her left nostril each morning upon arising. As soon as feasible it is planned to close the fistula by a plastic procedure through an intracranial operation.

Since the original draft of this paper was written, the patient has become pregnant again, giving further evidence of continued normal pituitary function.

SUMMARY AND CONCLUSIONS.

A brief review of the literature concerning the Arnold-Chiari malformation, together with report of a case, has been presented. From this review we conclude that some patients with this condition will be seen first by the otolaryngologist, with symptoms referable either to the labyrinth or other ear, nose and throat structures. Although the treatment may not be within the scope of our specialty, we should be aware of the possibility of the presence of this deformity.

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JOINT RESPONSIBILITY OF THE OTOLARYNGOLOGIST AND THE INTERNIST IN REMOVAL OF FOCAL OF INFECTION.*†

PAUL S. RHOADS, M.D. (by invitation),
Chicago, Ill.

Waning of the immense enthusiasm for removal of all accessible tissue suspected of harboring pathogenic bacteria has occurred for a variety of reasons. Failure, consistently, to demonstrate "elective localization" with bacteria cultured from suspected foci was one reason—though hardly a valid one. Others were: *1.* failure of patients with systemic disease to get well in a great number of instances after foci of infection apparently had been removed; *2.* the observation that many persons having chronically inflamed tonsils, infected sinuses, infected teeth, etc., did not at the same time have any systemic malady of the type usually attributed to such foci; *3.* failure to find obvious foci of infection in patients with long-standing rheumatoid arthritis; subacute bacterial endocarditis, nephritis, etc.; *4.* failure completely to remove foci of infection. We found tonsillar remnants in 72 percent of young adults who had previously undergone tonsillectomy.[2]

While the excesses in removal of suspected foci often practiced in the first two decades of this century are deplored, it is obvious that the arguments just cited against the importance of focal infection do not hold water.

Who can deny that bacteria growing in thrombi at the roots of infected teeth or in the bottom of the crypts of infected tonsils are frequently the cause of subacute bacterial

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†From the Department of Medicine, Wesley Memorial Hospital, and Department of Medicine, Northwestern University Medical School, Chicago, Ill.

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endocarditis? Who can deny that the rash of scarlet fever, diphtheritic paralysis and diphtheritic myocarditis are remote effects of the toxins produced by bacteria flourishing in a localized focus—as Billings[1] and his group contended?

Modern medical literature is filled with suggestive evidence that an even more important effect of continued low grade infection is the sensitization of certain tissues of the body in a way imperfectly understood so that they hyper-react to superimposed infection, giving rise to the so-called collagen diseases. Rheumatic fever is the outstanding example of this group of systemic affections; others are disseminated lupus erythematosis, periarteritis nodosa, dermatomyositis and probably acute nephritis. In a comprehensive review of the attempts through the years to induce rheumatic fever in experimental animals, Murphy[3] concludes that although the disease as it is seen in humans has not been successfully produced, lesions very similar to Aschoff bodies have developed in a certain proportion of animals after repeated focal cutaneous infections with group A streptococci. He points out that *single* cutaneous infections and infections due to a single intravenous injection of live culture, while often causing death of an animal, did not result in the typical lesions mentioned above. Why *repeated* focal infections caused myocardial, epicardial and vascular lesions in some animals and not in others just as happens with humans has not been satisfactorily explained. The observation of Murphy and Swift[4] that hypertrophy of the adrenal cortices was present in the animals developing rheumatic-like lesions while those failing to develop the Aschoff lesions did not show this change in the adrenals is of extreme interest.

Thomas[5] has shown that after intracutaneous inoculation of a live culture of hemolytic streptococci into the skin of a rabbit the intravenous injection of bacterial toxin into the animals results in a large area of hemorrhagic necrosis at the infected skin site and often carditis and bilateral renal cortical necrosis (generalized Shartzman phenomenon). The heart lesions bear a marked similarity to those of human rheumatic fever. That this sensitization is nonspecific is shown by the

fact that animals prepared by single, or better, multiple, focal infections with hemolytic streptococci will exhibit the generalized Shartzman reaction when a variety of bacterial toxins, including those of meningococci, are injected intravenously. Stetson[6] has made similar observations.

Why some humans, just as some laboratory animals, can harbor continuing low grade infections over long periods without developing systemic disease is poorly understood but does not invalidate the evidence against foci of infection acting as so-called tissue sensitizers. On the other hand, failure to observe swift amelioration of the symptoms of systemic disease after removal of focal infections does not prove that the focus was not involved in the entire phenomenon any more than failure of the plugging of a hole in a leaky boat to remove the water that has accumulated in the hull proves that the hole was not responsible for the accumulation of water in the first place.

RECOGNITION OF THE INFECTION.

Recognition of the infection in the upper portions of the respiratory tract in patients with chronic disability with or without low grade fever often taxes the diagnostic resources of both otolaryngologist and internist. Often a painstaking history and complete assay of the patient's physical condition are required to suggest, first, that a focus of infection must exist somewhere and, second, that it is probably in the nose, throat, inner ear or upper bronchi.

It is not the purpose of the present discussion to review the fine points in speculum examination of the nose and ears, examination of the nasopharynx or larynx with a mirror, etc. In all of these respects the otolaryngologist is more skilled than the internist.

There are, however, a few points to which I would direct your attention.

1. Is there evidence of allergic sensitivity in the patient's mucous membranes? If the patient's local complaints of intermittent obstruction to the breathing space in the nose, post-

nasal drainage, frequent sneezing and intermittent mild sore throat are of considerable duration and associated with very slight or no fever, certainly allergy is to be suspected. Associated asthma, chronic bronchiectasis or bronchitis make the likelihood even greater. The finding of a high proportion of eosinophiles in smears of nasal secretions, sputum or blood; seasonal variations in symptoms; and positive skin reactions to common allergens constitute strong presumptive evidence. It is important to recognize this allergic element, which is present in practically all cases of persistent rhinitis, sinusitis and pharyngitis so that the responsible allergic offenders such as dust, feathers, molds, pollens, foods, etc., may be discovered and eliminated as largely as possible from the patient's environment. Desensitization over long periods may have to be carried out. Antihistaminics will be helpful, nasal polyps may have to be removed, mucous membranes may have to be shrunk down to effect drainage, sinuses may have to be irrigated, large collections of lymphoid tissue blocking drainage may have to be removed from the nasopharynx in these allergic individuals.

2. Is there evidence of bacterial infection of the nose and throat? The simple procedure of taking the patient's temperature would save many persons from suffering indefinitely from low grade infections of the nose and throat if it were done on every visit because it would cause the physician to look further for infections in patients with elevated temperatures. It is doubtful whether nonbacterial allergy alone ever causes patients to develop fever.

Leucocytosis with a predominance of polymorphonuclears points toward a bacterial infection of the mucous membranes or lymphoid tissue, for which antibiotics or sulfonamide therapy is indicated. Counts of 12,000 to 16,000 are not uncommon in the absence of fever, and usually indicate bacterial infection. If the count is normal or low in the face of elevated temperature a virus infection is to be suspected. If there is a shift to the left in the differential count infectious mononucleosis or leucemia are to be considered. The Kahn or Wassermann blood tests are indispensable aids in clinching the

diagnosis when secondary syphilis is suspected; a positive Paul-Bunnell serologic test confirms the diagnosis of infectious mononucleosis.

What are the bacteriologic findings? In cases of severe sore throat, especially if an exudate is present, or if there is *purulent* rhinitis or draining sinusitis stained smears from material obtained by swabbing should be done. The presence of streptococci or pneumococci or H. influenzae in smears from the nasal secretions or aural discharge are definite evidence of infection. Pneumococci in large numbers in the throat or large numbers of spirilli and fusiform bacilli usually indicate a pathologic condition. Of course diphtheria bacilli are frequently found in direct smears also.

Cultures from the same locations also should be made on blood agar. When there is exudate on tonsils or pharynx or nasal mucosa or when there is evidence of laryngeal obstruction Loeffler's medium should be inoculated as well. Our own work does not support the idea held by some that hemolytic streptococci may, during certain seasons of the year, be benign parasites in the throat and nose. If they are present in considerable numbers in either throat or nose one may be reasonably certain that the patient is suffering or convalescent from an acute bacterial infection.[7] We made cultures of the noses and throats of medical students and nurses at different times throughout the year. When those found to harbor hemolytic streptococci were examined the majority were found to have low grade fever, leucocytosis and definitely inflamed mucous membranes even though they were not off duty. Nonhemolyzing streptococci in the throat or sputum cultures may be pathogenic, but since they are universally present in cultures from these areas they cannot be assumed to be pathogens.

Nonhemolytic staphylococcus albus usually is present in both nose and throat and practically never assumes pathologic significance. Hemolytic staphylococcus aureus, if it constitutes practically the total flora of the throat, may be a pathogen. If coagulase positive and associated with frankly purulent lesions it should be suspected of being of etiologic

significance. Friedlander's bacilli pseudomonas aeruginosa and B. proteus likewise may be harmless parasites, but if present in considerable numbers, especially following penicillin or aureomycin therapy, may be pathogens.

What are the physical findings? Purulent secretions from highly inflamed nasal mucous membranes or an intensely inflamed and painful throat, with or without exudate, are perfectly definite signs of infection—especially when associated with fever and leucocytosis. When these signs are present, cultures usually will reveal an abnormal bacterial growth, but even if nothing unusual is found, absence of infection cannot be assumed. In 1928, Dr. Dick and I[2] made quantitative cultures by grinding excised tonsils to a pulp with sand and broth in a sterile mortar. Most of the tonsils were not acutely inflamed at the time of operation, but we recovered hemolytic streptococci in every instance, whether or not they had been present in cultures made from the throat prior to operation. Pathogens may grow in abundance in the depths of tonsillar crypts without appearing on the surface.

When there is only moderate reddening of the mucous membranes, little or no tenderness over paranasal sinuses, little diminution in light transmission, no clouding on X-ray and no fever or leucocytosis, the problem of recognizing infection in the nasopharynx and paranasal sinuses becomes difficult. A chain of slightly enlarged lymph nodes back of the sternocleidomastoid muscle is rather definite evidence of infection in the nasopharynx, enlargement of the anterior cervical lymph nodes goes more often with tonsillar infection. Of course, such glandular enlargements may result from infectious mononucleosis, in which case one or two nodes stand out considerably larger than the others. In rheumatic fever almost invariably the posterior triangles of the neck are filled with small shotty glands which have been present so long that their presence may have been forgotten or ignored.

Attention to all the points enumerated should make it relatively easy to determine whether infection is present or not.

ELIMINATION OF THE INFECTION.

This brings us to the final point of this discussion. Having determined that an infectious process in the nose and throat, ear or upper bronchi is present, what is to be done? We are all probably pretty much in agreement on the course to pursue in the acute stage. If a bacterial infection due to hemolytic streptococci or pneumococci is present in nose, throat or ear, penicillin is doubtless the antibiotic of choice. Our personal preference is for the depot type in which crystalline is mixed with procaine penicillin—usually in the proportion of one to three, making a total dose of 400,000 units. This preparation administered intramuscularly every 12 hours, or 800,000 units every 24 hours, is adequate to control most streptococcal and pneumococcal infections. For those who prefer to use penicillin in the oral form it must be remembered that only 20 to 30 per cent of this antibiotic is absorbed from the intestine; hence the total dose must be four to five times as large as is used intramuscularly and the intervals between doses should be every three to four hours, day and night.

Aureomycin or terramycin in doses of 0.5 gm. every six hours, day and night, by mouth after an initial dose of 1 gm. may give equally good results.

In mixed infections or those in which the bacterial population is unknown, one of the broad spectrum oral antibiotics or a combination of dihydrostreptomycin with penicillin are the antibacterial agents of choice. If broad spectrum antibiotic coverage of these types is used over long periods the ascendency of microorganisms usually present as harmless saprophytes and not sensitive to the antibiotics being administered is a constant threat. These include K. pneumoniae, pseudomonas aeruginosa, B. proteus and sometimes staphylococci. The speed with which the latter acquire resistance to penicillin is disturbing. It is estimated that 60 per cent or more of staphylococci from the nose and throat are more resistant to penicillin. If an infection is considered to be due

primarily to staphylococci, titrations to determine the most effective antibiotic should be performed. These will usually prove aureomycin or erythromycin to be the ones of choice.

Sulfa drugs have been as impressive as the antibiotics in controlling infections of the nose, throat and ear—but they undoubtedly have a field of usefulness in persons who develop sensitivities to the antibiotics. If sulfa mixtures are to be used, those containing sulfadiazine, sulfamerazine and sulfathiazol are probably best and safest.

Whether antibiotics should be used locally in the nose and throat is a matter of individual judgment. It should be emphatically stated that unless the infection with which one is dealing is one of minor virulence this local treatment should be used only as an adjuvant, never as the sole treatment of the infection. Preparations containing mild vasoconstrictors such as 0.5 to 1 per cent solutions of paradrine, neosynephrine, etc., together with bacitracin, polymyxin B, tyrothricin or neomycin have rightly attained some popularity. All of the above preparations have a wide antibacterial range of effectiveness but are potentially too toxic for parenteral administration except under exceptional circumstances. They may be used safely as topical applications up to 500 units per ml. Having had an extensive experience with penicillin dust administered through Krasno's inhalator, I feel safe in saying that it is quite effective in combating sinusitis due to hemolytic streptococci and pneumococci providing the nasal passages are open. It is also useful in preventing the secondary bacterial invaders that so often follow in the wake of the common cold. It should never be used *in place* of parenteral therapy but usually in addition to it.

All of the measures which have just been discussed are conservative ones that may be administered by either internist or otolaryngologist. Operative interference or the local applications of antibacterial agents to remote recesses constitute the peculiar field of the otolaryngologist. The striking decrease in the necessity for paracentesis of the eardrum and for mastoidectomy illustrates the fact that many foci of infec-

tion can now be cleared up without surgical interference. When acute tonsillitis and purulent rhinitis are treated vigorously with antibiotics such conditions do not develop into chronically infected foci.

In the era before antibiotics the quarantine period for scarlet fever was four weeks. Our studies[10] revealed that 63 per cent of scarlet fever patients still had hemolytic streptococci in cultures of the nose or throat when dismissed from the hospital. With total daily doses of penicillin usually averaging less than 200,000 units daily, we reduced the carrier to 17 per cent as compared to 71 per cent in a control group of scarlet fever patients not so treated.[11] Only a few quantitative cultures of excised tonsils made by grinding them into a pulp in sterile sand and broth have been made in the past year in penicillin-treated patients. Hemolytic streptococci have been found in about half of these tonsils. This is bacteriologic evidence supporting the clinical impression that even deep seated infections may be cleared up by vigorous antibiotic therapy in many individuals.

Every one of us, however, has repeatedly observed chronic tonsillitis, suppurative otitis media, and suppurative sinusitis with accompanying lymph node involvement which improved to some extent while antibiotics were being administered, but promptly regressed upon their withdrawal. Almost always failure to effect a permanent cure has been due to inadequate drainage of some closed infected cavity—just as antibiotic therapy fails permanently to cure chronic urinary tract infections unless all obstructions to the free flow of the urinary stream are corrected. Antibiotic therapy alone will not free us from the responsibility of removing nasal polyps, shrinking down the mucosa of the turbinates, occasional irrigations of antrums which are the site of chronic suppuration, opening of tympanic membranes when there is frankly purulent exudate behind them, and other procedures to effect drainage of closed infection sites.

Lillie,[12] Williams[13] and Simonton,[14] of the Mayo Clinic, have devised a useful classification of chronic suppurative otitis

media and outlined the steps beyond antibacterial therapy to be taken for each type. The decision as to when mastoidectomy should be performed is a difficult one and must be the joint responsibility of otolaryngologist, Roentgenologist and internist. Suffice it to say that in spite of antibiotics this operation must occasionally be undertaken.

There was a time when tonsillectomy was the most frequently performed of all operations in the United States.[15] Fortunately that time has passed; however, there still are many patients, chiefly children and young adults, for whom tonsillectomy is indicated. When a young person has repeated bouts of tonsillitis with continuing evidences of chronic infection such as fever, leucocytosis, increased sedimentation rate, mild muscle and joint pains, cervical adenitis after the acute attack has subsided and vigorous antibiotic therapy has failed to clear up the infection, the indication for tonsillectomy seems clear. The tonsils of such persons will usually be found to be chronically inflamed, though not necessarily enlarged, with redness extending to the surrounding structures. Removal of such tonsils before rheumatic fever, rheumatoid arthritis, chronic infections of the urinary tract, subacute bacterial endocarditis, etc., develop is the most constructive thing to do. Improvement in these conditions from tonsillectomy *after* they have developed cannot always be expected, but it does seem rational to remove whatever active infection one can get at. It is hardly necessary to state that antibiotic therapy should precede operation for 24 hours and follow it for a few days. Of course, tonsillectomy should never be undertaken during the acute phase of rheumatic fever; however, in several instances when fever has continued several months after the joint pains and acute signs of carditis have subsided, we have seen the fever subside promptly after chronically diseased tonsils were removed.

Caylor and Dick[16] were the first to adopt the total quantitative bacterial count of excised tonsils as the best criterion of their harmful potentialities. They showed that, in general, tonsils with the highest bacterial content were those present in patients who had rheumatic fever, rheumatoid arthritis

and other systemic conditions associated with focal infection, while those with the lowest counts were hypertrophied tonsils removed because of local disturbances in the throat unassociated with systemic disease.

Rhoads and Dick[2] extended these studies, showing that tonsil stumps left from previous attempts at complete tonsillectomy had an average higher bacterial content per gram than tonsils removed for the first time. In such tonsillar remnants it was common to find much fibrosis and areas of necrosis at the base of crypts which had been sealed over at the surface as the result of the first operation. Sometimes infected thrombi were seen in the vessels draining the bottom of these crypts. In addition to tonsillar remnants they found a high bacterial count in small, smooth fibrous tonsils about which extension of the redness into the surrounding tissues was apparent. Microscopically the peritonsillar tissue or "capsule" which was stripped off when the tonsils were enucleated was found to contain collars of leucocytes about the blood vessels.[17] Machlachlan and Richey[15] were the first to point out the common association of such tonsils with rheumatic fever and rheumatoid arthritis. The other tonsils having high bacterial counts were those in which chronic lucunar tonsillitis with small abscesses on the tonsillar surface and in the crypts was the outstanding pathologic finding. Chronic hyperplastic tonsillitis was the diagnosis in the majority of those tonsils removed simply because the tonsils were enlarged and there was a history of repeated sore throats. Such tonsils had relatively low bacterial counts as compared to the others described above.

SUMMARY.

An attempt has been made to show that in spite of newer methods of treatment of nose and throat infection, a certain proportion of them progress to a localized chronic infectious focus, which is responsible in part, at least, for such systemic conditions as rheumatic fever, rheumatoid arthritis, iritis, bronchiectasis, chronic anemia and acute and subacute nephritis. Vigorous administration of antibiotics in the acute stage and even in the subacute or chronic stage has greatly

diminished the number and severity of such foci of infection. Recognition of the allergic nature of many of the chronic pathologic processes in the nose and throat will lead to methods of treatment that may make surgical interference unnecessary. When such foci exist, however, in spite of a combined antibacterial and antiallergic attack, they often must be removed surgically under the protection of administration of antibiotics.

Prevention of these foci by not allowing infections to become chronic and removal of them if they do develop is the joint responsibility of internist-otolaryngologist.

NOSE

NORMAL FLORA	PATHOLOGIC FLORA
STAPHYLOCOCCUS ALBUS	STREPTOCOCCUS VIRIDANS
STAPHYLOCOCCUS AUREUS	HEMOLYTIC STREPTOCOCCUS
STAPHYLOCOCCUS CITREUS	PNEUMOCOCCUS
B. PROTEUS VULGARIS	C. DIPHTHERIAE
SARCINAE	K. PNEUMONIAE
B. SUBTILIS	B. INFLUENZAE
	H. PERTUSSIS
	M. TUBERCULOSIS

THROAT

NORMAL FLORA	PATHOLOGIC FLORA
STAPHYLOCOCCUS AUREUS	HEMOLYTIC STREPTOCOCCUS
STAPHYLOCOCCUS ALBUS	C. DIPHTHERIAE
STAPHYLOCOCCUS CITREUS	H. INFLUENZAE
STREPTOCOCCUS VIRIDANS	H. PERTUSSIS
N. CATARRHALIS	
D. PHARYNGIS SICCUS	
B. SUBTILIS	
G. TETRAGENA	

QUESTIONABLE

STREPTOCOCCUS VIRIDANS IN PURE CULTURE
PNEUMOCOCCUS
FUSIFORMIS DENTIUM IN LARGE NUMBERS
BORRELIA VINCENTI IN LARGE NUMBERS
K. PNEUMONIAE
PSEUDOMONAS AERUGINOSA
M. TUBERCULOSIS

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INDICATIÓNS FOR TONSILLECTOMY AND ADENOIDECTOMY IN CHILDREN.*†

G. O. PROUD, M.D.,
Kansas City, Kan.

Statistics, submitted by a hospitalization insurance company in a certain metropolitan community in 1951 stated that the company paid 1,264 claims for maternity cases. The same organization reimbursed 1,311 claimants for tonsillectomy during the same period. Although it would be unscientific to infer from these data that there are in general more tonsillectomies than deliveries today, one should be struck by the realization than the operation is performed with amazing frequency. Supposedly, tonsillectomy is the most abused surgical procedure of this era, and it is tragic that this should be so, for physicians in the nonsurgical specialties have been forced to adopt an ultraconservative philosophy regarding diseases of lymphoid tissue. With the advent of antimicrobial therapy, operations for removal of lymphoid tissue have diminished in number; nevertheless, certain specific indications for surgical intervention remain, and we as specialists will do well to give pause for their consideration.

INDICATIONS FOR ADENOIDECTOMY.

Perhaps the most common indication for adenoidectomy is obstruction to nasal respiration. Mouth-breathing, the chief symptom, may be more apparent at night. In other patients, difficulty in eating may be a more prominent sign. Only the rare case presents the picture of adenoid facies. Palpation of the nasopharynx is distasteful to the patient, but only this procedure will give the physician assurance that the end-result of surgery will be less so.

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†From the Department of Otorhinolaryngology of the University of Kansas School of Medicine.

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Recurrent attacks of acute otitis media may cease after adenoidectomy, especially if lateral band dissection and removal of the "tubal bud" are also carefully carried out. Failures are more apt to ensue if the respiratory membrane is in a state of autonomic imbalance or if surgery is incomplete. It is surprising that adenoidectomy, when performed for the eradication of tubal disease, is so frequently successful. Despite the number of palate retractors and "round-the-corner" devices which have been introduced, the procedure is essentially a blind one. Division of the soft palate will at least provide a measure of manual and visual access to the nasopharynx which is not otherwise available.

Persistent subacute or chronic otitis media may resolve only after removal of the adenoid, particularly if the perforation occupies the so-called "tubal area." Here again, the difficulty may persist if the job is incompletely done, or if the origin of the disease lies in autonomic dysfunction or irreversible disease in the middle ear membrane itself.

The restoration of hearing following adenoidectomy for conductive type deafness in children or adults may be spectacular even in the absence of tympanic membrane change, and resection of the lateral bands seems to be of equal importance. If cases are carefully selected, failures are unusual but disappointing when they occur. Certain patients with otosclerosis probably come to needless adenoidectomy, but it is a greater misfortune when those with tubal deafness are subjected to the fenestration operation.

Recurrent or persistent secretory otitis media may be relieved by adenoidectomy if the causative factor lies in Eustachian irritation or obstruction rather than autonomic imbalance. Myringotomy may be carried out while the patient is still asleep and may give added assurance of resolution. Other reasons why adenoidectomy may fail to benefit secretory catarrh were recently enumerated by Suehs.[1]

It is most gratifying to see the measure of relief which adenoidectomy affords the occasional patient with vasomotor

rhinitis. If the obstruction is due in part to enlargement of the adenoid, its removal may make the individual comfortable in spite of the nasal edema. One would be unwise to advocate adenoidectomy in every case of vasomotor rhinitis, and only repeated examinations will determine just to what degree the adenoid is contributing to the obstruction.

Today adenoidectomy is frequently and justifiably done as an entirely independent procedure without tonsillectomy as an encore. Children so managed have minimal discomfort and few postoperative complications.

INDICATIONS FOR TONSILLECTOMY.

When abnormally large tonsils are removed, symptoms such as dysphagia and respiratory distress may be deleted.[2] It is occasionally surprising, however, to observe the total absence of discomfort of any sort in children with huge tonsils. In short, tonsillar hypertrophy of moderate amount is not an indication for surgery.

Quinsy, though rare in children, should be prevented by tonsillectomy, for subsequent recurrence is most likely. The respiratory embarrassment which may accompany a later attack may be alarming.

Tonsillectomy may be performed to halt recurrent Vincent's infection of the tonsil or to free an individual of the carrier state of diphtheria. The latter example is well discussed by Baglione.[3]

The rôle of the tonsil in the causation of acute nephritis and rheumatic fever is much in dispute. Perhaps it is well to advise tonsillectomy only in those instances in which the attacks of rheumatic fever or nephritis are antedated by acute tonsillitis.

Tonsillectomy may be a final necessary step in the eradication of a branchial cleft cyst, but the operation is most frequently done to prevent the recurrence of attacks of acute tonsillitis. It seems needless to mention that one or two mild attacks of tonsillitis are insufficient indication for surgery, but

in children who have unduly frequent or unusually severe episodes, the operation should be carried out. In this regard it is well to consider the so-called "ideal age" for tonsillectomy. The parent is often advised that the age of five years is perfect for tonsillectomy, for the youngster is now nicely able to tolerate a general anesthetic and should be "gotten ready for school." Pediatricians and laryngologists alike have come to realize that a child between the ages of four and 14 passes through a stage wherein the metabolic rate of his lymphoid tissue is greatly accelerated. At this time the lymphoid tissue is singularly vulnerable to disease. For example, youngsters in the upper limits of this bracket (ages 12 to 14) are on occasions subjected to appendectomy when in reality they are victims of acute mesenteric lymphadenitis. Younger children (ages four to 14), on the other hand, are prone to have recurrent bouts of tonsillitis and mysterious attacks of generalized adenopathy. Tonsillitis occurring during such a siege may simply be part and parcel of a generalized lymphoid tissue infection and not necessarily the cause of it.

Thus, it follows that most tonsillectomies will take place after age four and prior to age 12. This is by no means an ironclad dictum, for some children will enter this period of life earlier than others or leave it later. The tonsillectomy, in short, is best done when trouble begins, regardless of the patient's age.

CONCLUSIONS.

1. Although antimicrobial therapy spares some children the necessity of tonsillectomy or adenoidectomy, certain definite indications for one or both of these procedure remain.
2. The performance of adenoidectomy alone is justified in selected cases.
3. The ideal age for tonsillectomy is the age when trouble begins.
4. Failure to obtain a result from either operation may arise from incomplete surgery, faulty diagnosis or the presence of intercurrent disease.

5. Only careful postoperative follow-up of patients over many years will teach us the true value of our present-day indications.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

March, 1953.

Audicon Models 400 and 415.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

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Aurex (Semi-Portable).
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Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
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PROGRAMS OF NATIONAL OTORHINOLARYNGOLOGICAL SOCIETIES.

THE AMERICAN LARYNGOLOGICAL ASSOCIATION ANNUAL MEETING, ROOSEVELT HOTEL, NEW ORLEANS, LA., APRIL 26-27, 1953.

Primary Plasma Cell Tumors of the Upper Air Passages — Clyde A. Heatly, M.D., Rochester, N. Y.

Mesenchyme of the Nose and Sinuses—Aubrey C. Rawlins, M.D., San Francisco, Calif.

Primary Amyloid Disease of the Larynx—Julius W. McCall, M.D., Cleveland, Ohio.

The Laminagraph as an Aid in the Diagnosis of Diseases of the Larynx—Leroy A. Schall, M.D., and A. S. MacMillan, M.D. (by invitation), Boston, Mass.

Clinical Significance of Laryngeal Keratosis as a Premalignant Lesion—F. J. Putney, M.D., and John J. O'Keefe, M.D. (by invitation), Philadelphia, Pa.

Carcinoma-in-Situ of the Larynx — Alden H. Miller, M.D., and Russell Fisher, M.D. (by invitation), Los Angeles, Calif.

Chordoma. Final Report and Re-evaluation of Treatment — Henry B. Orton, M.D., Newark, N. J.

Neurilemmona (Schwannoma) of the Larynx—L. Chester McHenry, M.D., Oklahoma City, Okla.

Hemilaryngectomy and Subtotal Laryngectomy with Immediate Skin Graft—Frederick A. Figi, M.D., Rochester, Minn.

Treatment of Hyperplastic Sinusitis—Francis W. Davison, M.D., Danville, Pa.

Pharyngeal and Laryngeal Phenomena of Vascular Origin — French K. Hansel, M.D., St. Louis, Mo.

Precision Bronchology. A Must in Thoracic Surgery—Robert E. Priest, M.D., Minneapolis, Minn.

Surgical Complications of Diverticulum of the Pharynx — Kenneth A. Phelps, M.D., Minneapolis, Minn.

Realism in the Surgery of the Tonsils and Adenoids—Charles D. Blassingame, M.D., Memphis, Tenn.

Recent Items on Anesthesia of Interest to Laryngologists—John Adriani, M.D. (by invitation), New Orleans, La.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., ANNUAL MEETING—ROOSEVELT HOTEL, NEW ORLEANS, LA.

PROGRAM

TUESDAY, APRIL 28, 1953

Time

9:00—Election of Candidates
Election of Nominating Committee
Induction of Newly Elected Fellows

9:30—**Presidential Remarks**—Francis E. LeJeune, M.D., New Orleans, La.

9:40—**Introduction of Guest of Honor**—Charles A. Thigpen, M.D., Montgomery, Ala.

SCIENTIFIC SESSION

9:50—1. **Low Tone Deafness from VIIIth Nerve Section** (with slides)—Harold F. Schuknecht, M. D. (by invitation), Chicago, Ill.; Richard Woellner, B. A. (by invitation), Chicago, Ill.

10:10—Open Discussion

Time

10:20—2. **Bronchogenic Carcinoma: The Role of the Bronchoscopist in Its Early Diagnosis**—Alton Ochsner, M.D. (by invitation), New Orleans, La.

No Discussion

11:00—3. **The Formation of a Hearing and Speech Center**—Gordon D. Hoople, M.D., Syracuse, N. Y.; Louis M. DiCarlo, Ph.D. (by invitation), Syracuse, N. Y.

11:30—Open Discussion

11:40—15-MINUTE RECESS

11:55—4. **The Role of Rhinoplasty in Rhinology**—Maurice H. Cottle, M.D. (by invitation), Chicago, Ill.

12:15—Open Discussion

12:25—5. **Laryngeal Stenosis—Corrective Plastic Procedure for Failures Following Bilateral Abductor Paralysis Surgery** (Motion Picture)—DeGraaf Woodman, M.D., New York, N. Y.

12:45—Discussion—Robert C. McNaught, M.D., San Francisco, Calif.; G. S. Fitz-Hugh, M.D., Charlottesville, Va.

WEDNESDAY, APRIL 29, 1953

9:00—**"In Memoriam"**

Business Meeting—Report of the Nominating Committee; New and Miscellaneous Business

SCIENTIFIC SESSION

9:30—6. **Carcinoma of the Oral Cavity: Treatment by Dissection in Continuity** (Motion Picture)—Hans von Leden, M.D. (by invitation), Chicago, Ill.

10:00—Open Discussion

10:10—7. **Anatomical and Functional Considerations in Temporal Bone Surgery**—Julius Lempert, M.D., New York, N. Y.

No Discussion

10:40—8. **Neurofibromas of the Larynx—Report of Cases** (Motion Pictures)—Frederick A. Figi, M.D., Rochester, Minn.; David B. Stark, M.D. (by invitation), Rochester, Minn.

11:00—Open Discussion

11:10—15-MINUTE RECESS

11:25—9. **The Identification and Clinical Significance of Large Phagocytes in the Exudates of Acute Otitis Media and Mastoiditis**—William T. K. Bryan, M.D., St. Louis, Mo.

No Discussion

11:50—10. **Transoral Arytenoidectomy for Bilateral Vocal Cord Paralysis**—Fred Z. Havens, M.D., Rochester, Minn.

Open Discussion

THURSDAY, APRIL 30, 1953

9:00—Election of Council

Five-Minute Council Meeting

Presentation of New Instruments

SCIENTIFIC SESSION

9:30—11. **The Selection of Hearing Aids as an Office Procedure**—Werner Mueller, M.D., Boston, Mass.

9:50—Open Discussion

10:00—Remarks by the Governor of Louisiana, Mr. Robert Kennon.

10:15—12. **Medical Problems**—Louis H. Bauer, M.D. (by invitation), President of the American Medical Association, Hempstead, N. Y.

No Discussion

Time

11:00—15-MINUTE RECESS

11:15—13. **Some Newer Observations on Nasal Cilia**—G. Edward Tremble, M.D., Montreal, Quebec

11:35—Open Discussion

11:45—14. **A Re-evaluation of Semon's Hypothesis**—Louis H. Clerf, M.D., Philadelphia, Pa.; William H. Baltzell, M.D. (by invitation), Philadelphia, Pa.

12:05—Open Discussion

AMERICAN OTOLOGICAL SOCIETY, INC., ANNUAL MEETING—ROOSEVELT HOTEL, NEW ORLEANS, LA., MAY 1, 2, 1953

FRIDAY MORNING, MAY 1, 1953, 9:30 A.M.

INTRODUCTION OF GUESTS

Announcements—Presentation of New Instruments

1—**Address of President**—Albert C. Furstenberg, M.D.

2—**Introduction of the Guest of Honor**

3—**The Glomus Jugulare, a Nonchromaffin Paraganglion, in Man**—Stacy R. Guild, Ph.D.

4—**Surgical Repair of Perforated Eardrums** (Motion Picture)—Howard P. House, M.D.

5—**New Tests for Cochlear Reserve in the Selection of Patients for Fenestration**—Mr. James F. Jerger (by invitation).

6—Business Meeting

FRIDAY AFTERNOON, MAY 1, 1953, 2:00 P.M.

1—**A Laboratory Assessment of Hearing Acuity for Voice Signals Against a Background of Noise**—K. M. Simonton, M.D.

2—**Experimental Observations on Sound Conduction in the Middle and Inner Ear**—H. G. Kobrak, M.D., Ph. D. (by invitation)

3—**Development and Adult Structure of the Cochlear (Round) Window and of Related Portions of the Otic Capsule**—Barry J. Anson, Ph.D.; Theodore H. Bast, Ph.D.

4—**Nystagmus as an Indicator of Brain Lesions**—Elizabeth Crosby, Ph.D. (by invitation)

SATURDAY MORNING, MAY 2, 1953, 9:00 A.M.

1—**Theoretical Considerations of the Transmission of Sound Vibrations from the Perilymph to the Organ of Corti**—A. C. Hilding, M.D. (by invitation)

2—**Ototoxicity of Dihydrostreptomycin and Neomycin**—Moses H. Lurie, M.D.; Joseph E. Hawkins, Jr., Ph.D. (by invitation)

3—**Tissue Culture Techniques for the Study of the Isolated Otic Vesicle**—Merle Lawrence, Ph.D. (by invitation); Donald J. Merchant, Ph.D. (by invitation)

4—Business Meeting

SATURDAY AFTERNOON, MAY 2, 1953, 2:00 P.M.

1—**An Experimental Study of the Influence of the Autonomic Nervous System of the Inner Ear**—J. H. T. Rambo, M.D. (by invitation); Dorothy Wolff, Ph.D. (by invitation)

2—**Unrecognized Cholesteatosis in Children**—Philip E. Meltzer, M.D.

3. **Intravenous Procaine Treatment for Ménière's Disease**—Edmund P. Fowler, Jr., M.D.

4—Title and Essayist to be announced later

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